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THE LABYRINTH

THE LABYRINTH

AN AID TO THE STUDY OF INFLAMMATIONS OF THE INTERNAL EAR

By

ALFRED BRAUN, M.D. AND ISIDORE FRIESNER, M.D.
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WITH FIFTY FIGURES IN THE TEXT AND
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FOREWORD

A work on the labyrinth, at a time when this subject occupies the centre of interest in the otological world, requires no apology.

Although our knowledge of the subject of labyrinthine inflammations is still in the developmental stage, and opinions which we hold to-day may be controverted to-morrow, yet, in order to have a groundwork upon which to base our further study of this fascinating subject, it is necessary for us all to become familiar with those basic truths regarding labyrinthine disease, which have thus far been established beyond doubt.

These truths we have attempted to present in our book. On those topics, regarding which there is still considerable difference of opinion, we have made no attempt to state all the opinions, since this would only lead to confusion in the readers' minds. We have confined ourselves to those hypotheses which seemed most plausible to us.

In the bibliography, we have made no attempt to mention every work which has been written on this subject, but have referred only to those publications which were used in the preparation of this work.

The illustrations, made by one of us, are original, with the exception of a few which are modified from other illustrations.

We wish to thank Dr. Wendell C. Phillips and Dr. T. Passmore Berens for the opportunity they have given us to study cases of labyrinthine disease in their

clinics, and for their helpful suggestions in the preparation of the book. Our thanks are due to our colleague, Dr. J. J. Thomson, for his kind criticism and painstaking care in reviewing the manuscript.

To the Rebman Company our thanks are also due for their liberality and care in producing this book.

ALFRED BRAUN, M.D., ISIDORE FRIESNER, M.D.

NEW YORK CITY.

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CHAPTER I

ANATOMY

The internal ear consists of a membranous labyrinth enclosed in a bony capsule. It comprises the *vestibule,* the three *semicircular canals,* and the *cochlea.*

THE STATIC LABYRINTH

The bony static labyrinth consists of a **vestibule,** and three **semicircular canals.** The vestibule is situated between the cochlea in front, and the semicircular canals behind. It lies partly internal to the upper posterior portion of the middle-ear cavity, and partly internal to the aditus.

It is separated from the middle-ear cavity by the horizontal portion of the facial canal, and by the foot-plate of the stapes, which fills in the oval window. Internally, it is separated by a very thin plate of bone from the internal auditory canal. It is separated from the cochlea, in front, by a dense plate of bone, which contains the first portion of the facial canal. Thus it is seen that the vestibule is surrounded on two sides by the facial canal, namely, on its anterior and outer sides. This explains the frequent involvement of the facial nerve in labyrinthine disease. Posteriorly, the vestibule is bounded by a thick layer of compact bone.

The vestibule can be entered, surgically, from two directions, antero-externally, through the oval window, and postero-externally, through the solid-angle between the semicircular canals.

The roof, floor, anterior and posterior walls of the vestibule, correspond approximately in position to the roof, floor, anterior and posterior walls of the fundus of the internal auditory canal. The roof and the floor of the vestibule correspond, also, approximately, in position, to the upper and lower margins of the oval window.

The vestibule is roughly ovoid in shape, measuring, according to *Politzer,* 5 to 6 mm. in length, 3 to 4 mm. in width, and 4 to 5 mm. in height. Its capacity is about one minim.

It is usually described as having six sides, but this is more or less arbitrary, as the sides merge into each other imperceptibly.

The vestibule has opening into it the five ends of the three semicircular canals, namely, the **superior,** the **posterior,** and the **external.** Each canal has a large or ampullated extremity and a small extremity. Each of the ampullated extremities opens separately into the vestibule. The small ends of the superior and posterior canals join before entering the vestibule, and the small end of the external canal has a separate opening.

If the vestibule is bisected by a frontal cut, all of the openings of the semicircular canals can be seen in the posterior half. From above downwards the openings are as follows (Fig. 1, Plate I):

1. The ampullated end of the superior canal.
2. The ampullated end of the external canal.
3. The common opening of the superior and posterior canals.
4. The small end of the external canal.

PLATE I.

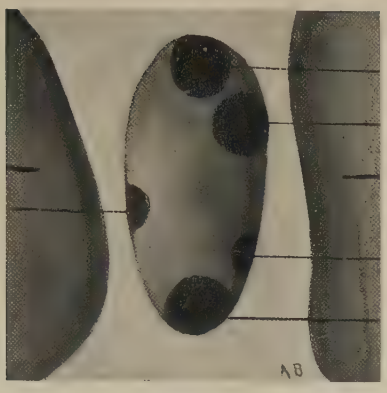

FIG. 1

CORONAL SECTION OF VESTIBULE OF LEFT EAR, SHOWING OPENINGS OF SEMICIRCULAR CANALS

(Looking at posterior half from in front)

ME—Middle ear
IAC—Internal auditory canal
SA—Ampullated end of superior canal
EA—Ampullated end of external canal
C—Common end of superior and posterior canals
E—Small end of external canal
PA—Ampullated end of posterior canal

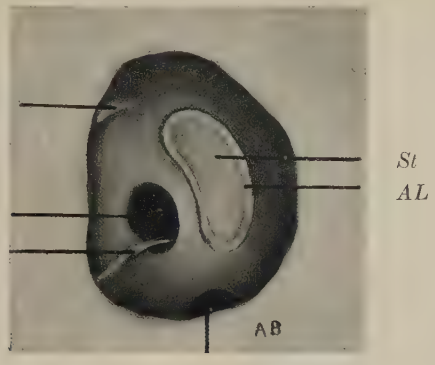

FIG. 2

OUTER WALL OF VESTIBULE (Right Ear)

St—Foot-plate of stapes in oval window
AL—Annular ligament
CV—Beginning of crista vestibuli
SV—Beginning of scala vestibuli
LS—Beginning of lamina spiralis
PC—Ampullated end of posterior semicircular canal

5. The ampullated end of the posterior canal.

The opening of the ampullated end of the superior canal lies at the junction of the roof with the posterior wall of the vestibule. The opening of the ampullated end of the external or horizontal canal lies just below and to the outer side of the ampullated end of the superior canal. The common opening of the superior and posterior canals lies at about the middle of the junction of the posterior with the internal walls of the vestibule. The opening of the small end of the external canal lies at the lower part of the junction of the posterior and outer walls of the vestibule. The ampullated end of the posterior canal lies in the floor of the vestibule.

The inner wall of the vestibule presents two shallow depressions separated by an oblique ridge, called the **crista vestibuli.** (Fig. 2, Plate I.) This ridge begins on the outer vestibular wall above the oval window, and runs from above and in front, downwards and backwards. The two shallow depressions are the recessus sphericus, which is situated in front of and below the ridge, and the recessus ellipticus, which is situated above and behind it. The recessus sphericus lodges the saccule, and the recessus ellipticus, the utricle.

The inner wall also contains, in the recessus ellipticus, just behind the crista vestibuli, a small foramen, the vestibular opening of the aqueductus vestibuli, which lodges the ductus endolymphaticus. It also contains a number of minute foramina, the maculæ cribrosæ, which transmit the fibres of the vestibular nerve. There are three sets of these foramina, the **macula cribrosa superior,** for the transmission of the utricular and the superior and external ampullary

nerves; the **macula cribrosa media,** for the transmission of the saccular nerve; and the **macula cribrosa posterior,** for the transmission of the posterior ampullary nerve.

The outer wall of the vestibule contains the oval window, which is filled in by the foot-plate of the stapes, surrounded by its annular ligament.

At the junction of the floor with the anterior wall of the vestibule is the opening into the cochlea. The vestibule communicates, at this point, with the upper division of the cochlea, the **scala vestibuli.** The beginning of the bony lamina spiralis of the cochlea is situated on the floor of the vestibule. (Fig. 3, Plate II.) The bony lamina spiralis, together with the membrana basilaris, shut off the scala tympani from the vestibule.

The bony semicircular canals are three in number. They are situated in the three planes of space, each at right angles to the other two. (Fig. 4.) There are two

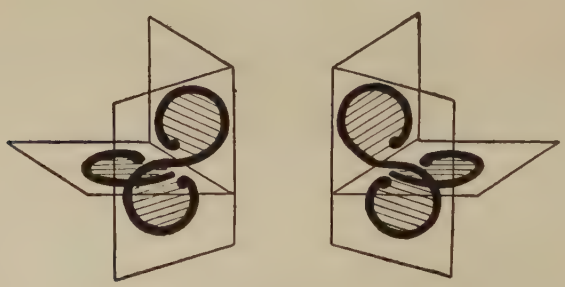

Fig. 4.

Planes of the Three Semicircular Canals, Modified After Ewald

vertical canals, the anterior vertical or **superior,** and the posterior vertical or **posterior.** The remaining canal is horizontal, and is usually called the

PLATE II.

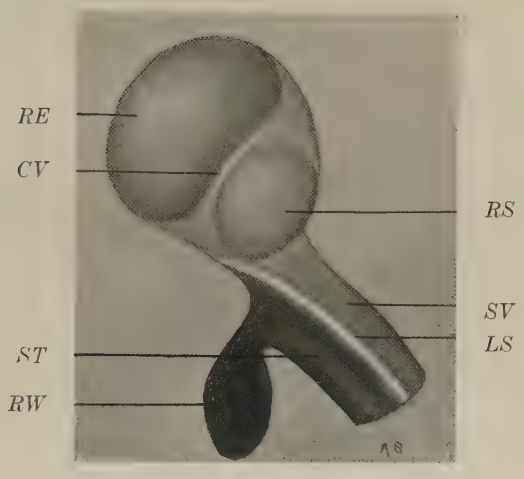

FIG. 3

INNER WALL OF VESTIBULE OF RIGHT EAR, AND BEGINNNING OF COCHLEA

RE—Recessus ellipticus
CV—Crista vestibuli
RS—Recessus sphericus
SV—Scala vestibuli of cochlea
LS—Beginning of lamina spiralis, showing its origin in floor of vestibule
ST—Scala tympani of cochlea
RW—Round window

external. It is best to call the canals, superior, posterior and external. The two vertical canals are sometimes called by the Germans, the frontal and sagittal canals. But these are not good terms, for the planes of these canals are not frontal nor sagittal, but form with these planes an angle of about 45°.

Schoenemann, from a large number of corrosion specimens, has come to the conclusion that the planes of the canals, as well as the angles between them, are very inconstant. The external canal is rarely horizontal. Its plane is tilted downward and backward, and forms with the horizontal plane an angle varying from 0° to 30°. This fact is important in interpreting the results of functional examination of the static labyrinth.

The angle which the plane of the superior canal makes with the medial plane varies between 30° and 65°. There may be a difference of 20° between the right and left sides. The angle between the superior and external canals varies between 65° and 90°. The angle between the external and posterior canals is most constant, varying from 90° to 100°, being usually 90°.

The superior canal of the right side is parallel to the posterior canal of the left, and the superior canal of the left side is parallel to the posterior canal of the right. (Fig. 5.)

Each semicircular canal comprises about two-thirds of the circumference of a circle, the diameter of which is 7 to 8 mm. They are elliptical on cross-section, and each canal has a slight enlargement at one end, called the ampulla. The canals are a little over 1 mm in diameter. The ampullæ are about 2½ mm in diameter. Both ends of each semicircular canal open into the ves-

tibule. The narrow ends of the superior and posterior canals join before they reach the vestibule, into which they open by means of a common limb. The ampulla of the superior canal lies at its outer end. The ampulla of the external canal lies at its anterior end. The ampulla of the posterior canal lies at its lower end. The ampullated ends of all three canals lie nearer to the middle ear than do the small ends. (Fig. 5.)

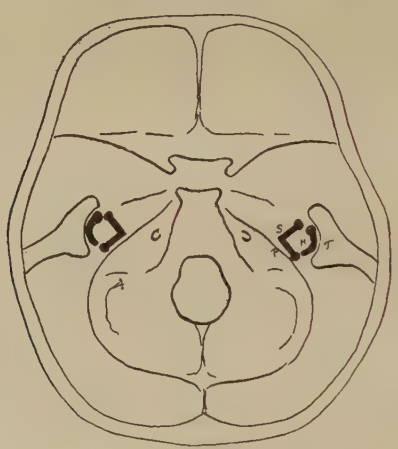

FIG. 5

PLANES OF THE THREE SEMICIRCULAR CANALS, WITH POSITIONS OF THEIR AMPULLÆ.

Interior of Skull, looking down from above

S—Superior semicircular canal
P—Posterior semicircular canal
H—External semicircular canal
T—Tympanic cavity

The **middle portion** of the **external semicircular canal** lies exposed in the attic and aditus. It forms a prominence on the inner wall of the attic and aditus, lying just above and behind the facial nerve, as it curves above and behind the oval window. In this situation, the external semicircular canal is very apt to be eroded by a suppurative process in the middle ear,

PLATE III.

FIG. 6

HORIZONTAL SECTION OF LEFT TEMPORAL BONE, THROUGH MIDDLE OF VESTIBULE (Lower half)

CC—Carotid canal
ET—Bony portion of Eustachian tube
T—Tympanic cavity
LS—Beginning of lamina spiralis
EAC—External auditory canal
FC—Facial canal
MA—Mastoid antrum
C—Cochlea
IAC—Internal auditory canal
V—Vestibule
PA—Ampullated end of posterior semicircular canal, in floor of vestibule
AV—Aqueductus vestibuli
E—Small end of external semicircular canal
P—Posterior semicircular canal

PLATE IV.

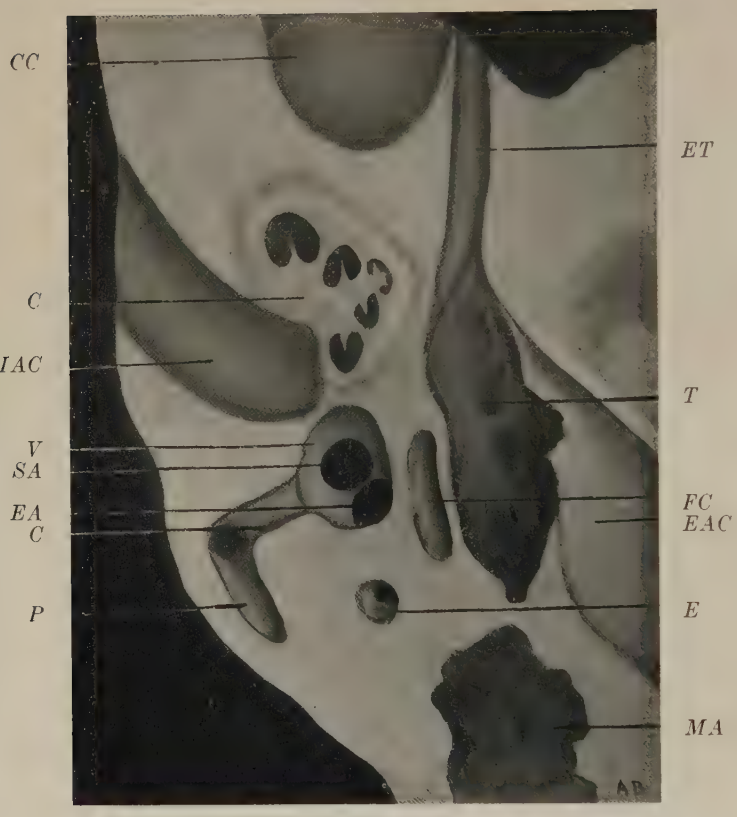

FIG. 7

HORIZONTAL SECTION OF LEFT TEMPORAL BONE, THROUGH MIDDLE OF VESTIBULE (Upper half)

CC—Carotid canal
C—Cochlea
IAC—Internal auditory canal
V—Upper half of vestibule
SA—Ampullated end of superior canal
EA—Ampullated end of external canal
C—Common end of superior and posterior canals
P—Small end of posterior canal, before it joins superior canal
ET—Bony portion of Eustachian tube
T—Tympanic cavity
FC—Facial canal
EAC—External auditory canal
E—External semicircular canal
MA—Mastoid antrum

PLATE V.

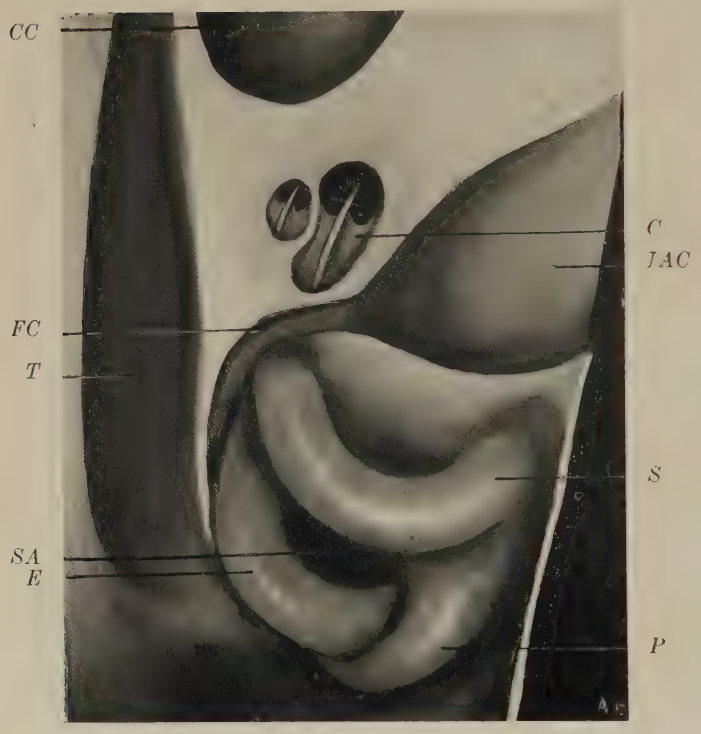

FIG. 8

DISSECTION OF LEFT TEMPORAL BONE, FROM ABOVE

Bony semicircular canals are dissected out, and cochlea, internal auditory canal and beginning of facial canal uncovered

CC—Carotid canal
FC—Facial canal
T—Tympanic cavity
SA—Solid angle
E—External semicircular canal
C—Portion of cochlea
IAC—Internal auditory canal
S—Superior semicircular canal
P—Posterior semicircular canal

and, as a matter of fact, this is the most common situation for a labyrinthine fistula.

The external semicircular canal is a guide in performing the simple mastoid operation, the radical mastoid, and in opening the vestibule.

In the simple mastoid operation, exposure of the external semicircular canal shows that the aditus has been reached. This marks the limit of the operation in that direction. In the radical mastoid operation, the external semicircular canal acts as a guide in determining how far it is safe to take down the posterior wall of the external auditory canal, without endangering the facial nerve, as the external semicircular canal projects outward slightly beyond the level of the facial nerve.

In opening the vestibule, the external semicircular canal acts as a guide, by marking the anterior limit of the bony excavation. If the chisel-cut passes in front of this canal, the facial nerve will be injured.

Injury of a normal external semicircular canal during the performance of a simple mastoid or radical operation is almost impossible, as it is covered by very dense hard bone. Yet occasionally it does occur.

The **superior semicircular** canal lies in a plane forming an angle of about 45° to the sagittal plane, passing from behind, outward and forward, the ampullated end being external and in front, and the inner and posterior end joining with the posterior canal to form the common limb. (Fig. 6, Plate III; Fig. 7, Plate IV; Fig. 8, Plate V.)

The superior canal forms a prominence on the upper surface of the petrous bone, called the **eminentia**

arcuata, beneath which, on the posterior surface of the bone, is a fossa, known as the fossa subarcuata. This lies a short distance above and to the outer side of the internal auditory meatus. It served, in the fetus, to transmit a large vein from the interior of the petrous bone. The prominence on the upper surface of the bone corresponds in shape to, but does not always actually represent the superior semicircular canal, inasmuch as the canal is frequently separated from the surface by a layer of cancellous bone, which is sometimes as much as a millimeter in thickness.

The superior canal is the furthest of the three from the mastoid antrum, and is the most difficult to reach in doing the labyrinth operation. The dangers in opening the superior canal are the possibility of injuring the dura of the middle fossa, the possibility of injuring the superior petrosal sinus, and the possibility of injuring the facial nerve, as it winds around the anterior extremity of the canal.

The **posterior semicircular canal** lies very close to the inner table of the posterior fossa, its convexity corresponding to a point on the posterior surface of the petrous bone, about half way between the inner opening of the aqueductus vestibuli and the inner margin of the groove for the sigmoid sinus. Its plane is parallel to the posterior surface of the petrous bone. It reaches down as low as the level of the round window. Its lower limb lies over the jugular bulb, and just internal to the descending portion of the facial nerve (Fig. 9, Plate VI). Its upper limb joins the superior canal, to form the common limb, and the lower ampullated end opens into the floor of the vestibule.

PLATE VI.

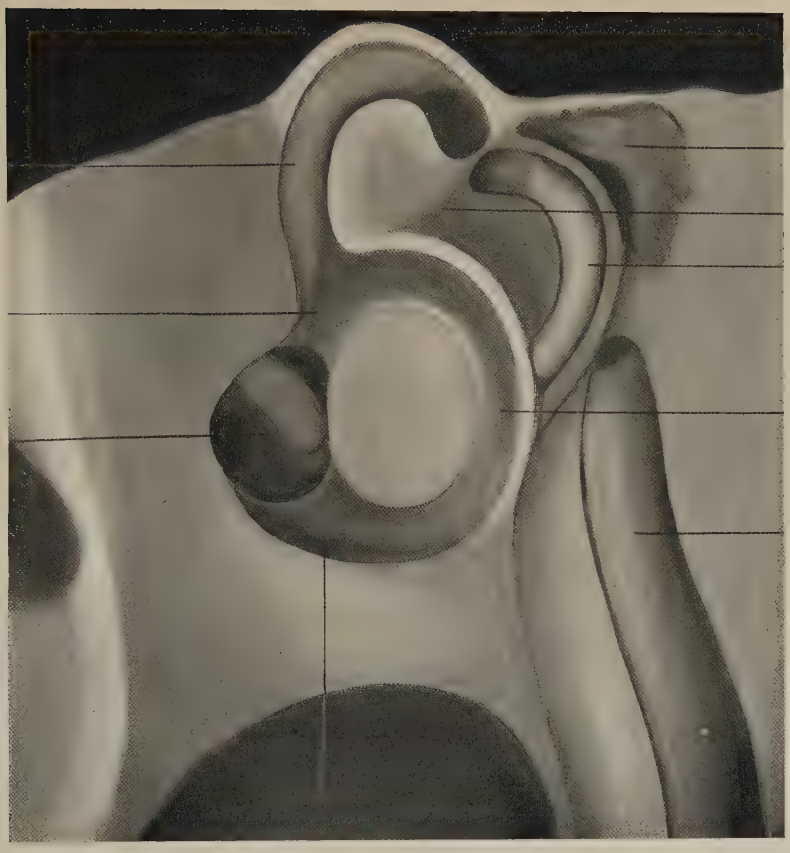

FIG. 9

DISSECTION OF SEMICIRCULAR CANALS FROM BEHIND (RIGHT EAR).

SC—Superior semicircular canal
CL—Common limb of superior and posterior canals
V—Vestibule, showing foot plate of stapes and beginning of scala vestibuli of cochlea
IAC—Internal auditory canal
A—Aditus
SA—Solid angle
EC—External semicircular canal
PC—Posterior semicircular canal
FC—Facial canal
APC—Ampullated end of post. sem. canal
JF—Jugular fossa

PLATE VII.

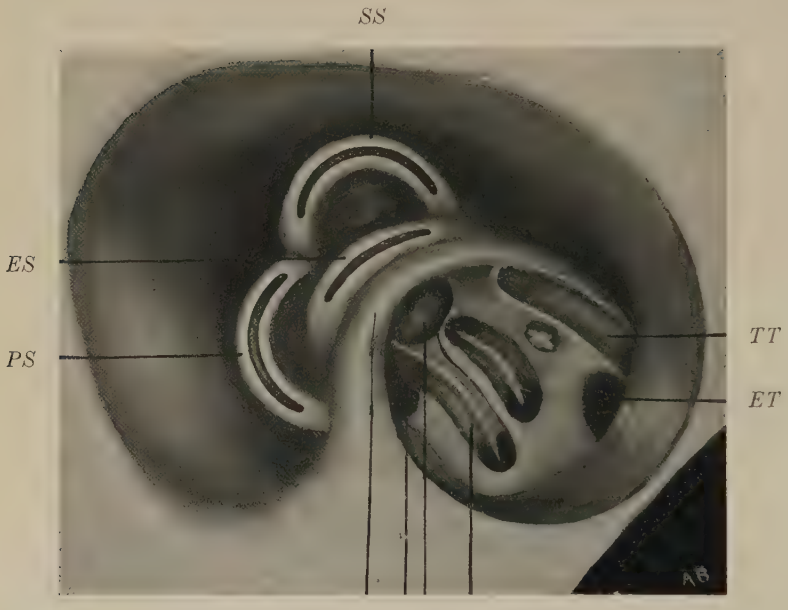

FIG. 10

RADICAL MASTOID OPERATION—COCHLEA AND SEMICIRCULAR CANALS OPENED (Right Ear)

SS—Superior semicircular canal
ES—External semicircular canal
PS—Posterior semicircular canal
FR—Ridge containing facial nerve
RW—Round window
V—Vestibule
C—Basal whorl of cochlea. The second and beginning of the third whorls are opened, but not marked
TT—Groove for tensor tympani tendon
ET—Tympanic orifice of Eustachian tube

The danger in opening the posterior canal consists in the possibility of injuring the cerebellar dura, the facial nerve, and the jugular bulb.

The angle between the three semicircular canals is composed of cancellous tissue, and is called the **solid-angle.** (Fig. 10, Plate VII.) By removing the bone in the solid-angle the vestibule can be entered from its posterior outer side.

The bony labyrinth consists of a layer of dense ivory-like bone, 2 to 3 mm. in thickness, which in the fetus and the new-born reaches the surface of the petrous bone in many places. In the adult, this capsule is covered over by spongy bone, and shows only at the promontory, the external semicircular canal, and, sometimes, at the superior canal. This dense bony structure is called the **labyrinthine capsule.** In chronic necrotic processes, the spongy bone about the labyrinthine capsule may be destroyed, and the whole or a large part of the labyrinth may be exfoliated as a single sequestrum.

The membranous static labyrinth lies within the bony labyrinth, to which it is attached by fibrous bands. It has, roughly, the same shape as the bony labyrinth.

The membranous labyrinth constitutes a closed fibrous tissue sac, which is not in communication with the endocranial lymph spaces. The fluid within the membranous labyrinth passes through the **ductus endolymphaticus** into the **saccus endolymphaticus,** which is a closed sac, lying between two layers of the dura, on the posterior surface of the petrous bone.

However, the space outside of the membranous labyrinth, between the membranous and bony labyrinths,

which contains the **perilymph,** is in direct communication with the subarachnoid space, through the **aqueductus cochleæ,** and the lymph spaces in the internal auditory canal.

The membranous portion of the static labyrinth consists of two sacs, the utricle and the saccule, the canalis utriculo-sacccularis, the ductus and saccus endolymphaticus, the canalis reuniens, and the three membranous semicircular canals and their ampullæ. (Fig. 11.)

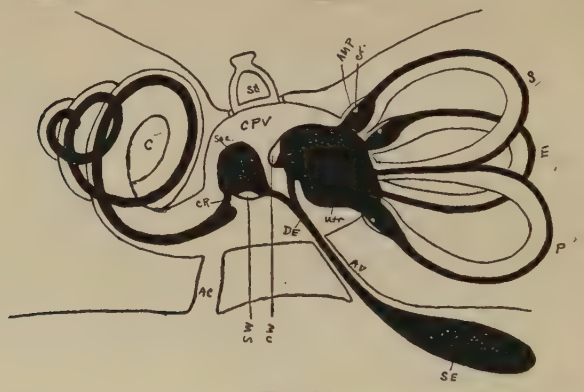

FIG. 11

ENTIRE BONY AND MEMBRANOUS LABYRINTH FROM BEHIND. RIGHT EAR (Schematic)

The membranous labyrinth is drawn in solid black

C—Cochlea
S—Superior semicircular canal
P—Posterior semicircular canal
E—External semicircular canal
AMP—Ampulla of superior canal
Cr—Crista ampullaris
St—Stapes in oval window
CPV—Cisterna perilymphatica vestibuli
Sac—Saccule
MS—Macula sacculi
Utr—Utricle
Mu—Macula utriculi lying in recessus utriculi
CR—Canalis reuniens
AC—Aqueductus cochleæ
AV—Aqueductus vestibuli
DE—Ductus endolymphaticus
SE—Saccus endolymphaticus

The **utricle** and **saccule** are two membranous sacs which lie within the bony vestibule. The utricle, the larger of the two, lies above and behind, in the re-

PLATE VIII.

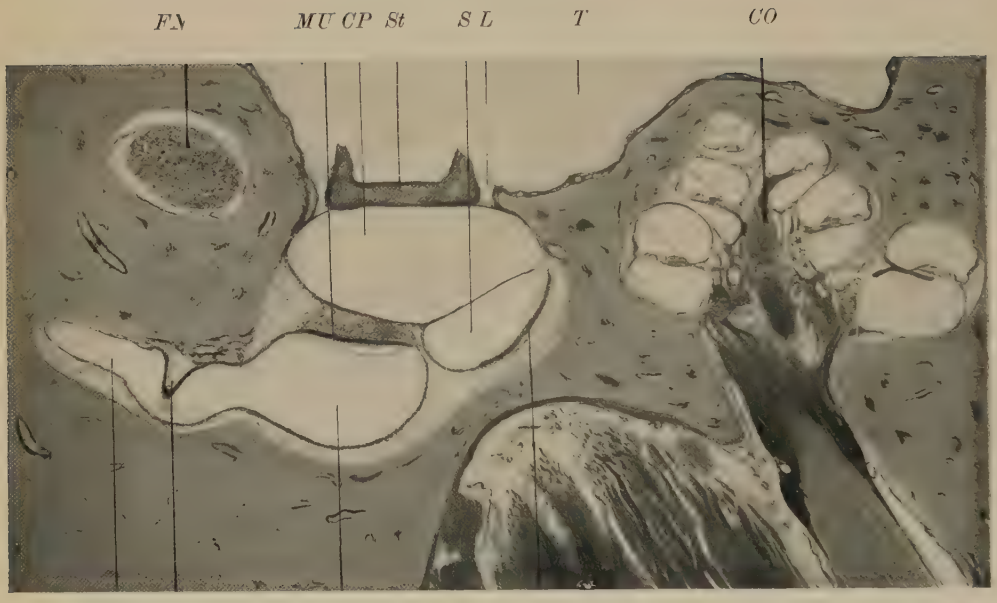

FIG. 12

HORIZONTAL SECTION OF INTERNAL EAR

CO—Cochlea
T—Tympanic cavity
L—Ligament of foot-plate of stapes
St—Foot-plate of stapes lying in oval window
CP—Cisterna perilymphatica vestibuli
S—Saccule
MS—Macula sacculi
U—Utricle
MU—Macula utriculi
HSC—External semicircular canal
CrH—Crista of external semicircular canal
FN—Facial nerve
VN—Vestibular nerve lying in internal auditory canal
CN—Cochlear nerve

cessus ellipticus, and the saccule, the smaller of the two, lies in front and below, in the recessus sphericus.

The utricle is an irregularly shaped sac, into which the three membranous semicircular canals open, by five openings. It is attached at its upper and inner surfaces to the bony vestibule. In its upper and anterior portion there is a pouch, called the recessus utriculi, which contains the nerve end-organ, the **macula utriculi.**

The saccule is oval in shape, with its long axis vertical. It is attached to the inner wall of the bony vestibule. It has two openings, one above and one below, the upper leading to the canalis utriculo-sacculari s, and the lower to the canalis reuniens. It contains a macule analogous to that in the utricle. The space between the outer walls of the utricle and saccule, and the outer wall of the bony vestibule, is filled with perilymph, and is called the **cisterna perilymphatica vestibuli.** (Fig. 12, Plate VIII.)

The two maculæ, according to *Breuer,* lie in planes at right angles to each other. The macule of the utricle lies on the antero-lateral aspect of the recessus utriculi, its base being turned to the cisterna perilymphatica. The **macula sacculi** lies on the medial wall of the saccule, against the bony inner wall of the vestibule.

According to *Breuer* the otoliths of the macula utriculi glide horizontally, from behind and external, in a direction forward and inward; while the otoliths of the macula sacculi glide vertically, from above and behind, in a direction downward and forward.

The saccule is connected to the ductus cochlearis, or membranous cochlea, by means of a short narrow canal,

the canalis reuniens. The saccule and utricle are joined together by a narrow canal, the canalis utriculo-saccularis, consisting of two limbs, which, by their junction, form another canal, the ductus endolymphaticus. The ductus endolymphaticus enters the aqueductus vestibuli through an opening on the inner vestibular wall, and, passing in a direction backwards and outwards, emerges through an opening on the posterior surface of the petrous bone, midway between the internal auditory meatus, and the inner edge of the groove for the sigmoid sinus. Here the ductus endolymphaticus spreads out into a sac-like expansion, the saccus endolymphaticus, which lies between two layers of the dura. The tip of the saccus endolymphaticus often reaches to the inner edge of the sigmoid sinus, and sometimes even lies in the medial wall of sinus. (Fig. 13.)

The membranous semicircular canals have only about one-eighth of the diameter of the bony canals, according to *Alexander*. The membranous ampullæ, however, are almost as large as the bony ampullæ. The membranous canals and ampullæ are attached to the convex surfaces of the bony canals.

The space about the membranous canals is called the **perilymphatic space,** and is continuous with the cisterna perilymphatica of the vestibule. The perilymphatic space is traversed by numerous connective tissue trabeculæ, which run from the membranous canals to the bony wall, and which are lined by endothelial cells.

Each of the membranous ampullæ contains a nerve end-organ, called the **crista-ampullaris,** which is an elevated ridge protruding into the lumen of the

ampulla, lying at right angles to the axis of the canal. The crista lies on that side of the ampulla, which is continuous with the convex surface of the bony canal. The crista divides each ampulla into two portions, a longer tubal portion, which is the side in the direction of the canal, and a shorter sinus portion, which is the side directed toward the utricle.

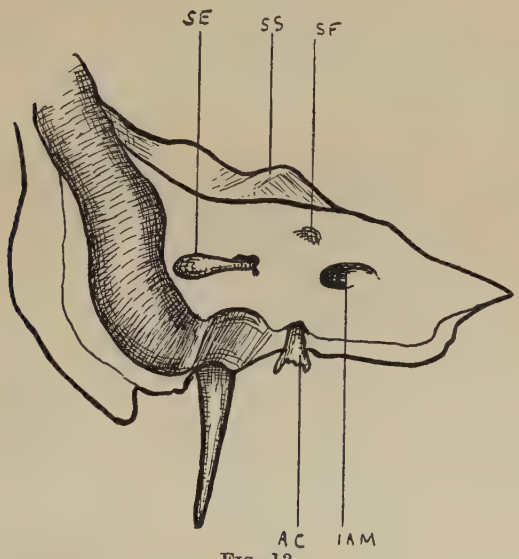

FIG. 13

POSTERIOR SURFACE OF TEMPORAL BONE, SHOWING SACCUS ENDOLYMPHATICUS AND AQUEDUCTUS COCHLEÆ

SE—Saccus endolymphaticus emerging from aqueductus vestibuli
SF—Fossa subarcuata
AC—Aqueductus cochleæ
IAM—Internal auditory meatus
SS—Eminence of superior semicircular canal

The membranous semicircular canals, the utricle and saccule, are all similar in histological structure. They are composed of three layers, an outer fibrous layer, a delicate basal membrane, and a single layer of squamous epithelial cells.

The **fibous layer** is similar to the periosteum lining the bony canals and vestibule, with which it is continuous, at the points where the membranous canals are attached to the bone.

The **basal membrane** is homogeneous and very

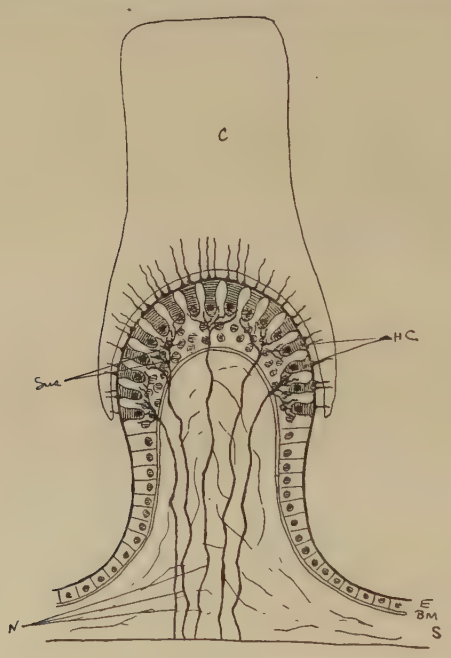

FIG. 14

CROSS-SECTION OF CRISTA AMPULLARIS (Schematic)

S—Submucosa
BM—Basement membrane
E—Epithelium lining ampulla
HC—Hair-cells
C—Cupula
Sus—Nuclei of sustentacular cells
N—Nerve fibres breaking up into network to surround bases of hair-cells

thin. It, as well as the connective tissue layer, becomes thicker at the maculæ and cristæ.

As we approach the maculæ and cristæ the squamous epithelium becomes columnar, and in the end-organs changes into neuro-epithelium.

The histological structure of the maculæ and cristæ is very similar. (Fig 14.) The cristæ are raised above the inner surface of the ampullæ, whereas the maculæ are flush with the inner surfaces of the utricle and saccule. The gelatinous membrane overlying the maculæ contains otoliths, while the membrane overlying the cristæ contains none. The end-organs consist of an outer fibrous layer, a basal membrane, and neuro-epithelium. The **neuro-epithelium** consists of two kinds of cells. The so-called fibre cells, which are supporting cells, are elongated and slightly expanded at

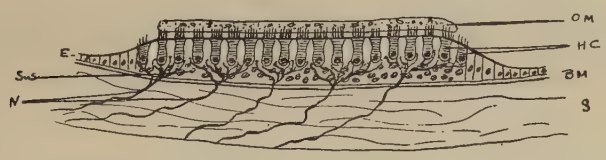

Fig. 15

Cross-Section of Macula Acoustica (Schematic)

S—Submucosa
BM—Basement membrane
E—Epithelium lining saccule and utricle
Sus—Nuclei of sustentacular cells
HC—Hair-cells
N—Nerve fibres breaking up into network to surround bases of hair-cells
OM—Membrane containing otoliths

the lower and upper ends, and traverse the entire thickness of the epithelium. They have an oval nucleus, situated below the level of the second type of cells, the hair-cells. The **hair-cells** are the real nerve end-organs. They occupy only the upper half of the epithelium. They are flask-shaped, with a lower rounded end, which contains a large spherical nucleus. (Fig. 15.)

From the surface of the hair-cells are given off long delicate hairs, 20 to 25 microns in length. These hairs enter the gelatinous membrane, which overlies the end-

organ. The membrane which overlies the crista is called the **cupula.** It is a clear, gelatinous membrane, which coagulates after death. It overrides the crista like a saddle. The hairs of the hair-cells penetrate this structure, and when the motion of the endolymph is imparted to it, all of the hairs move with it.

The membrane over the maculæ, which is called the **otolith-membrane,** is similar to the cupula, with the exception that it contains otoliths, which are small stones, 2 to 25 microns in diameter, composed of calcium carbonate and calcium phosphate.

The end-organs are supplied by the terminal filaments of the vestibular nerve. The nerve fibres, when they reach the basilar membrane under the neuro-epithelium, lose their medullary sheaths, and break up into terminal filaments, which ascend between the supporting cells to the bases of the hair cells, which they surround by means of a basket-like network.

Cajal claims that the nerves are not equally distributed to the hair-cells in the cristæ, the cells at the top of the crests being much more richly supplied by nerve fibres than those on the declivities.

Kolmer, Bruehl and *Bielschowsky* think that they have proven that the nerve fibrils terminate in the hair-cells themselves, whereas *Cajal* claims that they merely surround the cells. There are anastomoses between the nerve fibrils, and each nerve fibril supplies a large number of hair-cells.

THE COCHLEA

The cochlea is embedded in the petrous portion of the temporal bone, between the internal auditory canal and

the cavity of the tympanum. It is snail-like in shape, and consists of a canal 28 to 30 mm. long, which, starting from the vestibule, winds a little more than two and a half times around its axis. The turns, in their beginning, are directed from the vestibule behind and above, toward the carotid canal below and in front. The diameter of the canal, at the beginning, is about 2 mm., and gradually grows smaller as the canal approaches its superior extremity.

The cochlea is in close relation to two large blood-vessels. The basal whorl lies directly above the dome of the jugular bulb, while the internal carotid artery is in front of and external to the cochlea, and its canal is separated from the latter by only a thin layer of spongy bone.

The central structure of the cochlea, about which the canal winds, is known as the **modiolus.** This is conical, and begins with a broad base at the external end of the internal auditory canal, then taking a general direction outward, forward and upward, extends almost to the apex or cupola of the cochlea. The base of the modiolus is considerably thinner at its edges than it is at its centre. In the direction of the axis of the modiolus, from its base to its apex, there are numerous canals containing nerves and blood-vessels. In the centre of the modiolus the canalis centralis modioli runs from the base to the apex. In the lower part of the modiolus the axial canals, including the canalis centralis, are not completely filled by their contents, but as the canals approach the apex their walls lie closer to the contained vessels and nerves. *Richards* has shown that in uncapping the cochlea, the modiolus may be removed from

its apex toward its base, to a point corresponding to the termination of the first cochleal whorl, without causing the loss of cerebro-spinal fluid, i. e., without placing the operative field and intracranial contents into gross communication.

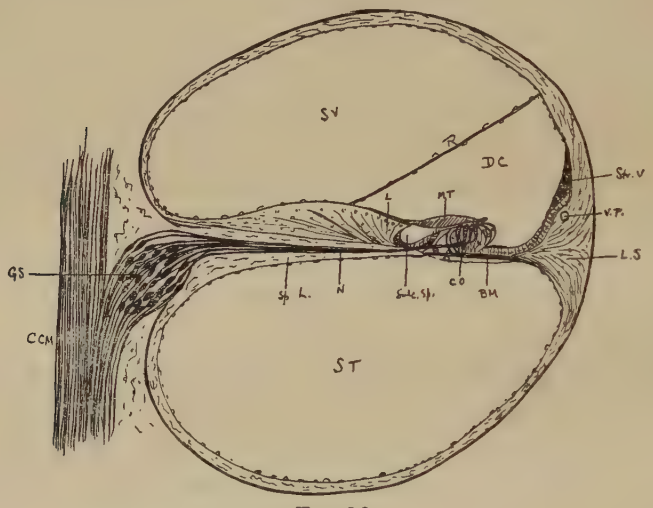

Fig. 16

Section Across Basal Whorl of Cochlea

ST—Scala tympani
SV—Scala vestibuli
DC—Ductus cochlearis
R—Membrane of Reissner
BM—Basilar membrane
LS—Ligamentum spirale
CCM—Canalis centralis modioli, containing cochlear nerve and vessels
GS—Ganglion spirale
Sp.L.—Bony lamina spiralis
N—Nerve fibres passing to Corti's organ
CO—Organ of Corti
MT—Membrana tectoria
L—Limbus
Sulc. SP—Sulcus spiralis
Str. V—Stria vascularis
VP—Vas prominens

Winding about the modiolus, on the outer surface, at its point of junction with the osseous lamina, is the **canalis spiralis** of *Rosenthal,* which contains the spiral ganglion. (Fig. 16.) Beginning at the lower limit of the oval window, and apparently continuous

with the floor of the vestibule, a thin plate of bone, the **lamina spiralis ossea,** winds about the modiolus, at right angles to it, partially dividing the canal of the cochlea. The outer edge of this bony plate gives attachment to the membranous spiral lamina, which, gradually becoming broader as it ascends, completes the division of the cochlear canal into a lower and an upper scala. The lower scala is called the **scala tympani,** and ends at the fenestra rotunda, where the membrana tympani secundaria separates it from the tympanic cavity. The upper is called the **scala vestibuli,** and communicates with the vestibule. The scalæ communicate with each other at the apex of the cochlea through the helicotrema Berschetti.

Two portions of the cochlea present in the inner tympanic wall. They are the promontory and the round window. The cochlea, however, runs considerably farther forward than the anterior boundary of the tympanum, and lies partly internal to the Eustachian tube, and partly internal to the carotid artery. The promontory represents the outer wall of the first cochleal whorl. This wall is usually thick and dense, but occasionally it is so thin that it is penetrated by the knife-blade in performing a paracentesis, thus establishing a communication between the infected middle ear and the labyrinth. The basal whorl may be indicated on the inner tympanic wall by a line drawn from a point on the posterior tympanic wall, midway between the oval and the round windows, downward and forward, to the junction of the anterior tympanic wall with the floor. This line forms an angle of about 45° with the hori-

zontal plane. The **round window** lies at the bottom of a recess situated at the middle of the posterior part of the inner tympanic wall. In looking at the latter through the external canal, one can see only the recess and not the round window itself, as the plane of the window is at right angles to the plane of the inner tympanic wall. The secondary tympanic membrane lies in the coronal plane. It is therefore impossible to injure it in doing a paracentesis. Infections from the cochlea may reach the intracranial contents by two routes; one through the internal auditory canal, along the sheaths of the nerves and vessels, and the other through the aqueductus cochleæ.

The **internal auditory canal** begins in a large orifice, the meatus auditorius internus, about the centre of the posterior surface of the pyramid. From the internal auditory meatus it runs obliquely outward and backward. It is in direct line with the external auditory canal, the two being separated from each other by the labyrinth and middle ear. In regard to its length and width, the canal varies considerably in different individuals. Its outer end is divided by a transverse shelf of bone into an upper and a lower fossa. In the anterior part of the upper fossa is an opening which marks the beginning of the facial canal, and transmits the facial nerve. In the posterior part of the upper fossa there are openings for the passage of some of the branches of the vestibular nerve. At the bottom of the lower fossa are the spirally arranged openings at the base of the modiolus, for the transmission of the divisions of the cochlear nerve. There are, besides, a few small openings on the posterior wall of the internal au-

ditory canal, through which some branches of the vestibular nerve pass.

As has been described, the canal of the cochlea is divided into two scalæ by the lamina spiralis ossea and the lamina spiralis membranacea, or membrana basilaris. The latter arises from the edge of the bony lamina, and extends to the outer wall of the cochlea, where it is attached to the ligamentum spirale, a vascular fibrous thickening of the endosteum. Extending from the upper surface of the osseous lamina, near its outer edge, to the ligamentum spirale, above the insertion of the membrana basilaris, is the **membrane of Reissner.**

This is a thin, homogeneous, radially striated membrane, containing occasional spindle-cells. By means of the membrane of Reissner, the scala vestibuli is divided into a large upper part and a smaller lower one. The lower section, called the ductus cochlearis, is the endolymph space of the cochlea, and contains the terminal filaments of the cochlear nerve. The ductus cochlearis is triangular on cross section, bounded above by the membrane of Reissner, externally, by the **ligamentum spirale,** and below, by the **membrana basilaris.** It is lined by a layer of flattened epithelium, and terminates near the cupola, above, and at the round window, below, in rounded blind extremities. Its only communication is with the saccule, by means of the delicate **canalis reuniens.**

The scala tympani, and the large upper division of the scala vestibuli, are the perilymph spaces of the cochlea. They communicate with each other at the

apex of the cochlea by means of the helicotrema. The scala vestibuli is continuous behind with the vestibule. Just beyond the fenestra rotunda, in the floor of the scala tympani, an opening leads to a fine canal, which extends to the inferior surface of the petrous bone near its posterior border, where it communicates with the subarachnoid space. The canal is called the **aqueductus cochleæ,** and affords an avenue of communication between the perilymph sac and the subarachnoid space.

On the upper surface of the bony spiral lamina, between its free edge and the point of origin of Reissner's membrane, there is a thickening of the periosteum, that has a toothed ridge. From this ridge there projects outward a gelatinous structure of relatively considerable thickness. This is the **tectorial membrane,** and it runs outward almost to the outermost point of the organ of Corti, and lies on the upper surface of the latter like a thick cushion, the hairs of the hair-cells projecting into or against it (*A. A. Gray*). (Fig. 17.) The membrana tectoria is analogous to the gelatinous cupula of the crista ampullaris, and the otolith membrane of the macula. Beneath it, is the organ of Corti, which may be considered as composed of five parts, viz.:

1. Sense epithelial cells, or hair-cells.
2. Sustentacular cells (rods of Corti, and the cells of Deiters).
3. The membrana tectoria, already described.
4. A reticular membrane which overlies the hair-cells, and is connected with the sustentacular cells.
5. The endings of the auditory nerve fibres.

The **hair-cells** are columnar in shape, with rounded bases. The lower part of the cell contains the nucleus, and is in contact with the terminal nerve-filaments. The hair-cells form four or five rows. These are arranged in two series, an inner and an outer, and are separated from each other by the rods of Corti. The inner series consists of a single row, and is next to the spiral lamina. The outer series consists of three or four rows. The hair-cells of Corti's organ differ from

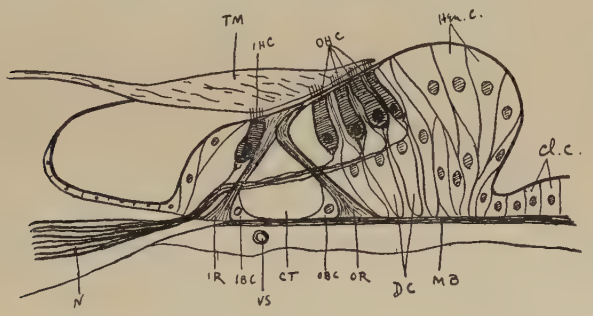

FIG. 17.

SECTION OF ORGAN OF CORTI (Schematic)

IR—Inner rod
OR—Outer rod
CT—Tunnel of Corti, with nerve-fibres passing across
IBC—Inner basilar cell
OBC—Outer basilar cell
MB—Membrana basilaris
TM—Tectorial membrane
N—Nerve-fibres
VS—Vas spirale
IHC—Inner hair-cell
OHC—Outer hair-cells
DC—Deiters' cells
Hen. C—Hensen's cells
Cl. C—Claudius' cells

those of the maculæ in that the cilia of the former are comparatively short and are arranged in the form of a crescent on the free surface of the cell. According to *Scott,* the cilia are gathered together in bundles like sheaves of corn, and pass down into the substance of the cells.

Separating the inner and outer rows of hair-cells are

the **rods** of **Corti.** These form a double row of peculiarly shaped cells, which, starting far apart, in broad bases, on the membrana basilaris, incline toward each other as they rise, and finally meet at their apices. They enclose a triangular passage, the tunnel of Corti. They rise stiffly from the membrana basilaris, and from their arrangement, are evidently supporting cells. To their upper free ends the reticular membrane is attached. Lying between the outer hair-cells are the **cells** of **Deiters.** These also rise stiffly from the membrana basilaris to the reticular membrane, and with the latter and the rods of Corti form a protecting framework for the hair-cells. The rods of Corti and the cells of Deiters are analogous to the supporting cells of the crista ampullaris. Beyond the outer hair-cells the epithelium (cells of Hensen) gradually changes in form, becoming cylindrical (cells of Claudius), and finally cuboidal. The latter type is continued along the membrana basilaris to the ligamentum spirale. Internal to the inner row of hair-cells, the epithelial cells gradually become shorter, until they merge imperceptibly into the flattened epithelium lining the ductus cochlearis.

The reticular membrane is a net-like structure, connected with the free ends of the rods of Corti and the cells of Deiters. Through its apertures project the hairs of the hair-cells. These extend to or into the soft gelatinous membrana tectoria.

The nerve fibres of the cochlea are derived from **bipolar cells** which form a spiral ganglion at the outer surface of the modiolus, at its junction with the spiral bony lamina. The fibres then pass through radially arranged channels in the latter, and enter the

organ of Corti, near the bases of the hair-cells. As the nerve leaves its bony channel it loses its sheath; only the axis-cylinder enters the organ of Corti. Some fibres end at the inner row of hair-cells, others, piercing the tunnel enclosed by the rods of Corti, pass to the outer hair-cells. *Kishi* believes that the nerve fibres which pass to the outer hair-cells lie on the floor **(membrana basilaris),** and not above it, as they pierce the tunnel of Corti. The central axons of the bipolar cells, in the spiral ganglion, pass through the channels which have been described in the modiolus, and are then collected to form the cochlear division of the auditory nerve.

The cochlea receives its blood-supply through the **internal auditory artery,** a branch of the basilar. According to *Siebenmann,* this artery sends but a single twig into the labyrinth itself. The remaining branches supply the bony capsule, the roots of some of the cranial nerves, and part of the under surface of the cerebellum. This single labyrinth artery divides into three main branches.

1. **The vestibular artery,** which accompanies the vestibular nerve, sends branches to the upper part of the saccule and utricle, and to the ampullæ of the superior and external semicircular canals. Small vessels follow the canals along their concave surfaces.

2. **The cochlear artery,** which winds in the canalis spiralis, and here, according to *Böttcher,* sends capillary branches into the ductus cochlearis.

3. **The vestibulo-cochlear artery,** which supplies part of the cochlea, the lower part of the vestibular structures, and the ampulla of the posterior

canal. *Politzer* has shown that an anastomosis of the labyrinthine with the tympanic vessels occurs through the medium of vessels traversing the outer bony labyrinthine wall.

The veins of the labyrinth are collected into three trunks. One forms a rich plexus in the internal auditory canal, a second follows the aqueductus vestibuli, while the third accompanies the aqueductus cochleæ. All three empty into the inferior petrosal sinus.

THE EIGHTH NERVE AND ITS CENTRAL CONNECTIONS

The auditory, or better the eighth nerve, arises from the medulla by two large roots, a mesial, or **radix vestibularis,** and a lateral or **radix cochlearis.** The nerve thus formed lies external to the facial, and passes forward, in company with the latter, to the internal canal. From the common trunk of the eighth nerve, a filament is given off, which supplies the ampulla of the posterior semicircular canal. The remainder of the nerve bundle is divided into two principal branches, a lower, which enters the cochlea, and an upper, which is further subdivided. The upper division, the utriculo-ampullaric, runs, at first, in common with the facial, then separating from it, passes through the inner wall of the vestibule, to its anterior surface, where its deep fibres supply the utricle, and are distributed ultimately to the macula utriculi; its superficial fibres supply the ampulla of the superior semicircular canal, and a lateral

bundle passes to the ampulla of the external canal. Between the two principal branches of the eighth nerve the filament which supplies the saccule is given off. The vestibular ganglion is common to both the saccular and the utriculo-ampullaric nerves, at their point of entrance into the bone, and it follows the utriculo-ampullaric branch a short stretch into the bone. *Winkler* showed that ganglion cells are scattered along the course of the vestibular nerve, and that the vestibular ganglion in the guinea-pig is in close contact with the medulla.

Wittmaack, Marx and others proved, by section of the eighth nerve in the internal auditory canal, that despite complete loss of function of the static labyrinth, the vestibular nerve showed no decided degenerative changes, while the cochlear nerve showed a distinct descending degeneration throughout its entire extent, even to the neuro-epithelium.

As has already been described, the cochlear nerve has its large bipolar ganglion cells in the canalis spiralis. From this point the nerve fibres arise out of the ganglion cells and pass through the radiating canals of the lamina spiralis, where they anastomose freely. Only the axis-cylinders pass out of the canals, and are distributed to the hair-cells of the organ of Corti. Whether the filaments enter the cells, or merely surround them, is still a mooted question.

The maculæ of the vestibular structures, and the cristæ of the canals, are supplied by the filaments of the vestibular branches. The nerve-fibres, when they reach the basement membrane under the neuro-epithelium, lose their sheaths, and break up into terminal

filaments, which, after a free anastomosis, are distributed to the hair-cells. Here also, the question as to whether they enter the cells, or are distributed in a basket-like formation about their bases, is still unsettled.

With regard to the important rôle which the eighth nerve plays in the extension of labyrinthine suppuration to the intra-cranial contents, *Lange* states: "The eighth nerve, at its entrance into the internal auditory meatus, is surrounded by a more or less loosely adherent process of the arachnoid. This process runs free in the canal, unattached to the dural lining of the latter, until it reaches the fundus, at which point it becomes adherent to the wall. To the facial, this arachnoid covering is attached chiefly in front and above, and it follows the nerve for a short distance into its canal, where it merges with the lining of the latter. The sheath leaves the vestibular and cochlear nerves, however, sooner than it does the facial, and so attaches itself to the dura, as to enclose the entire areæ cribrosæ. With individual differences, there exists, in the depths of the internal auditory canal, a larger or smaller cavity, almost exactly funnel-shaped—an extension of the subarachnoid space—in which the nerve breaks up into its branches. Between the nerve fibres in the cochlea there are large lymph spaces, which also play an important part in the extension of inflammatory processes from within this organ. The subdural space has no communication with this extension of the subarachnoid space in the internal auditory canal."

Just as the peripheral fibres arise in the ganglia along the course of the eighth nerve, so, too, do the cen-

tral fibres, and while the former are distributed to the end-organs, the latter are gathered into bundles, which finally merge to form the two roots in which the eighth nerve arises. Of these two roots the radix cochlearis (lateral root) passes to the outer side of the restiform body, where it is enlarged by numerous nerve cells, and terminates about the cells of the ventral and dorsal nuclei of the eighth nerve, some of the fibres being apparently continuous with the auditory striæ on the floor of the fourth ventricle.

The central connections of the cochlear division of the eighth nerve, according to *Lewandowski,* are as follows (Fig. 18):

From the ganglion of the lateral root *GL,* and the ventral accessory nucleus *Vn* of the cochlear nerve, the fibres pass across the medulla and pons, to the opposite side, principally, through the trapezium *Tr* to the nucleus of the trapezium *TrN,* through *Held's* acoustic fibres *H* to the upper olive *UO,* and through the acoustic striæ *St,* to the upper olive. Some of the fibres pass directly to the nucleus of the lateral fillet *NLF.* A few fibres do not cross over, but pass up through the lateral fillet of the same side.

From the upper olive, and the nucleus of the trapezium, fibres pass through the lateral fillet *LF* and its nucleus *NLF,* to the posterior corpus quadrigeminum, *PCQ.*

Through the brachium of the posterior corpus quadrigeminum *BCQ,* fibres pass to the internal geniculate body *ICB,* and from here they pass to the cortex of the temporo-sphenoidal lobe *C.*

There is a commissure *P,* described by *Probst,* be-

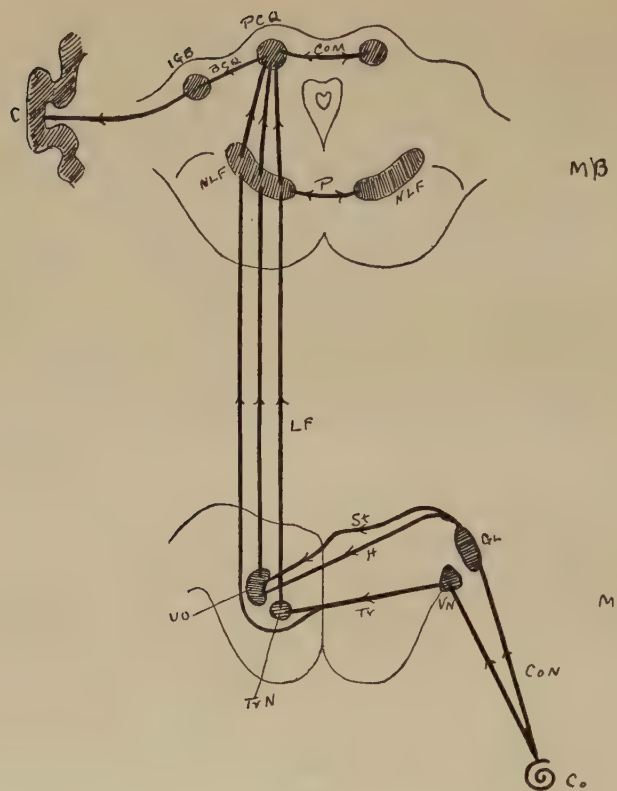

FIG. 18

CENTRAL CONNECTIONS OF COCHLEAR NERVE. (Modified after Lewandowsky)

(The uncrossed fibres are not indicated, for the sake of clearness)

M&P—Junction of medulla and pons
MB—Mid-brain
Co—Spiral ganglion of cochlea
CON—Cochlear nerve
VN—Ventral accessory nucleus of eighth nerve
GL—Ganglion of lateral root
Tr—Trapezium
TrN—Nucleus of trapezium
H—Held's acoustic fibres
St—Striæ acousticæ
UO—Upper olive
LF—Lateral fillet
NLF—Nucleus of lateral fillet
P—Probst's commissure
PCQ—Posterior corpus quadrigeminum
COM—Commissure between posterior corpora quadrigemina
BCQ—Brachium of posterior corpus quadrigeminum
IGB—Internal geniculate body
C—Cortex of temporo-sphenoidal lobe

tween the two nuclei of the lateral fillet, and a commissure *COM* between the two corpora quadrigemina. Through the commissures, as well as through the few fibres of the secondary auditory tracts which do not cross to the opposite side, each cerebral cortex is brought into communication with both ears.

The radix vestibularis (mesial root) passes obliquely backwards on the inner side of the restiform body to its central nuclei in the floor of the fourth ventricle. It has not, as yet, been possible to isolate and follow separately the sacculo-utricular and ampullaric elements of the vestibular root. *Kölliker* and *Cajal* have shown that the fibres, in their course to the central nuclei behind the descending root of the trigeminus, divide into ascending and descending limbs, forming with the trunk a letter *Y*.

According to *Kohnstamm,* the following are the central nuclei of the vestibular root (Fig. 19):

1. Large-celled nucleus of Deiters.
2. Small-celled or ventro-caudal nucleus of Deiters.
3. Nucleus triangularis.
4. Nucleus angularis, or Bechterew's nucleus.
5. Nucleus embolo-globosus.

From the other so-called vestibular nuclei the large-celled Deiters' is distinguished by the fact that by the *Marchi* method no vestibular fibres can be proven to enter it. These fibres, however, probably do act upon it through the intervention of the nucleus triangularis. Into the ventro-caudal or small-celled Deiters' nucleus vestibular fibres can be traced. This nucleus, gradually growing smaller as it descends, passes downward in the

medulla, along with the fibres of the descending root of the vestibular nerve, for a short distance.

The nucleus triangularis is in the floor of the fourth ventricle, and bears such a close relation to the nuclei of the vagus, that it is possible for an impulse to one

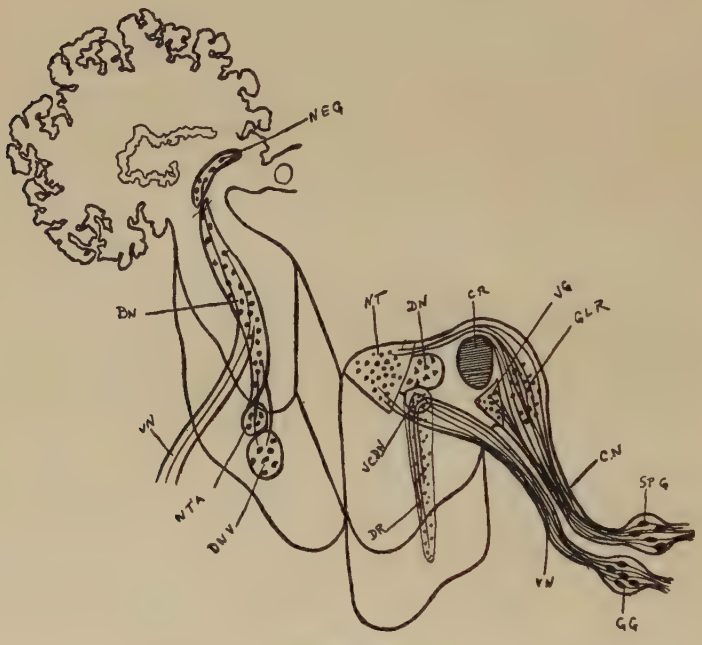

FIG. 19

NUCLEI OF EIGHTH NERVE (Schematic)

SpG—Spiral ganglion of cochlear nerve
GG—Geniculate ganglion of vestibular nerve
CN—Cochlear nerve
VN—Vestibular nerve
VG—Ventral accessory ganglion of cochlear nerve
GLR—Ganglion cells in lateral root
NT—Nucleus triangularis (Dorsal nucleus)
DN—Deiters' nucleus
VCDN—Ventro-caudal Deiters' nucleus
DR—Descending root of vestibular nerve
BN—Becheterew's nucleus (nucl. Angularis)
NEG—Nucleus embolo-globosus
NTA—Nucleus trigemino-angularis
DNV—Nucleus of the descending root of the fifth nerve
CR—Corpus restiforme

centre to be radiated to the other. This explains the nausea and vomiting which occur with vestibular vertigo.

Before the work of *Kohnstamm* and *Quensel* nothing was known regarding the function of the nucleus angularis (*Bechterew*). This nucleus extends dorsally, without any demonstrable dividing line, into the nucleus embolo-globosus of the cerebellum. Ventrally it extends into the nucleus of the descending spinal root of the fifth nerve, through the interposed bridge of the nucleus trigemino-angularis. (Fig. 19.) The nucleus angularis, the nucleus embolo-globosus and the nucleus trigemino-angularis are identical as regards the form of their cells, and in preparations, cannot be differentiated. They may really be regarded as parts of a single nucleus. In its degenerative changes, the nucleus trigemino-angularis resembles the sensory trigeminal nucleus more than it does the nucleus angularis (*Bechterew*). *Kohnstamm* states that for a long time he had observed what was published by *May* and *Horsley*, viz., that many fibres from the degenerated root of the fifth nerve can be followed into Bechterew's nucleus.

The nucleus embolo-globosus, which is intermediary between Bechterew's nucleus and the central nuclei of the cerebellum must also be considered among the vestibular nuclei. For degenerated fibres of the vestibular nerve can be followed to a point that is beyond Bechterew's nucleus, and within the nucleus embolo-globosus. Moreover, in injuries to the mid-brain, which are followed by widespread degenerative changes of Bechterew's nucleus, the nucleus embolo-globosus also shows tigrolysis.

By the Marchi method it is impossible to demonstrate completely the physiologic-anatomical relations of the vestibular nuclei. For in their positions these bear such complicated relationships to one another, that they cannot be affected singly through lesions artificially produced, and so the dependence of degenerated fibres upon one or another of the nuclei cannot be determined with absolute precision. However, in the vestibular nuclei the central tracts of the vestibular nerve begin. These have been demonstrated successfully by the Nissl method.

According to *Cajal,* the fibres of the ascending limb of the root of the vestibular nerve terminate in the cells of Deiters' nucleus and Bechterew's nucleus. The fibres of the descending limb terminate in the nucleus triangularis, Deiters' nucleus, and the ventro-caudal or accessory Deiters' nucleus, in the last of which the fibres exhaust themselves.

According to *Kohnstamm,* however, Deiters' nucleus does not receive any vestibular fibres directly, the impulses reaching Deiters' nucleus through the triangular nucleus.

From the vestibular nuclei secondary tracts pass down through the spinal cord and up through the pons and mid-brain. *Kohnstamm* was able to identify the following connections by the Nissl method in guinea-pigs (Fig. 20):

From Deiters' nucleus fibres pass down through the Deiterso-spinal tract of the same side. This tract passes through the antero-lateral tract of the spinal cord *Deit Sp,* sending collaterals to the anterior horn cells of the cord.

From the nucleus angularis (Bechterew's nucleus) fibres pass upward through the posterior longitudinal fasciculus *PLF*, in the posterior part of the reticular formation of the pons, and the tegmentum of the mid-brain, on both sides, the fibres being more numerous on the same side. In the mid-brain the fibres terminate about the cells of the nuclei of the nerves supplying the ocular muscles.

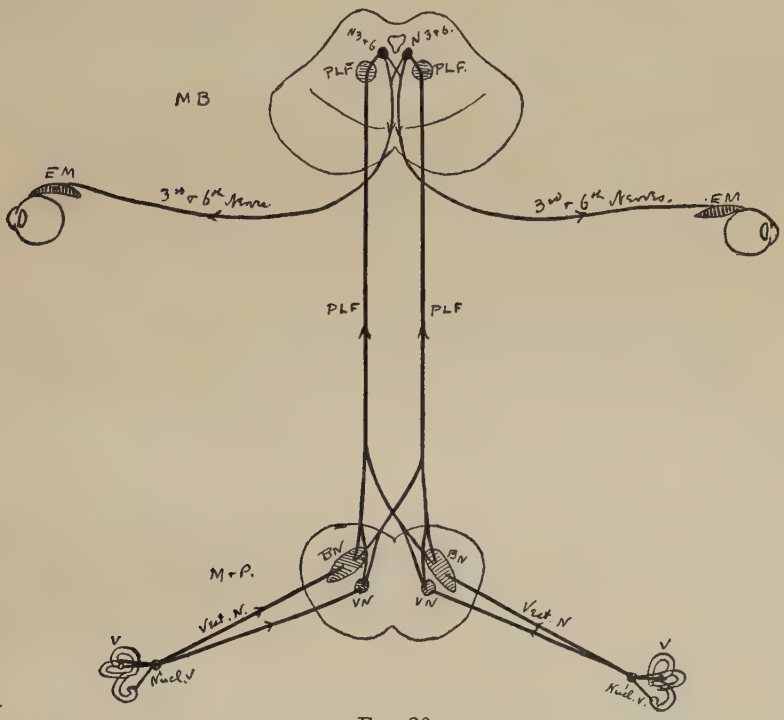

FIG. 20

TRACTS INVOLVED IN NYSTAGMUS

MB—Mid-brain
M & P—Junction of medulla and pons
V—Vestibule
Nucl. V—Vestibular nucleus
Vest N—Vestibular nerve
BN—Bechterew's nucleus
VN—Ventro-caudal Deiters' nucleus
PLF—Posterior longitudinal fasciculus
N3&6—Nuclei of third and sixth nerves
EM—Ocular muscles

From the ventro-caudal or accessory Deiters' nucleus, a few fibres pass down through the dorsal longitudinal bundle of the cord, to the anterior horn cells on both sides; and a few fibres pass upwards, through the posterior longitudinal bundle of the pons and mid-brain *PLF,* on both sides, to the nuclei of the nerves supplying the ocular muscles.

Consequently the reflex paths, through which the movements of nystagmus are intermediated, are as follows: From the vestibule, through the vestibular nerve, to the vestibular nuclei. From Bechterew's nucleus and the ventral accessory Deiters' nucleus to the posterior longitudinal fasciculus on both sides, to the nuclei of the nerves of the ocular muscles, and through the third, fourth and sixth nerves, to the ocular muscles.

The following are the pathways of reaction movements, according to *Barany:* The pathways for reaction movements of the extremities are different, in a portion of their course, from those of the body.

The impulses for reaction movements of the extremities pass from the vestibule through the vestibular nerve to Deiters' nucleus (Fig. 21, *Deit*), and the ventro-caudal nucleus *VD.* From here, through the corpus dentatum of the cerebellum *DN,* to the cortex of the cerebellar hemisphere. From the cerebellar cortex, fibres pass through the brachium conjunctivum to the red nucleus of the tegmentum of the opposite side *RN.* From here they pass down through Monakow's bundle *Mon* across to the lateral tract of the spinal cord, to the anterior horn cells of the opposite side. Thus the

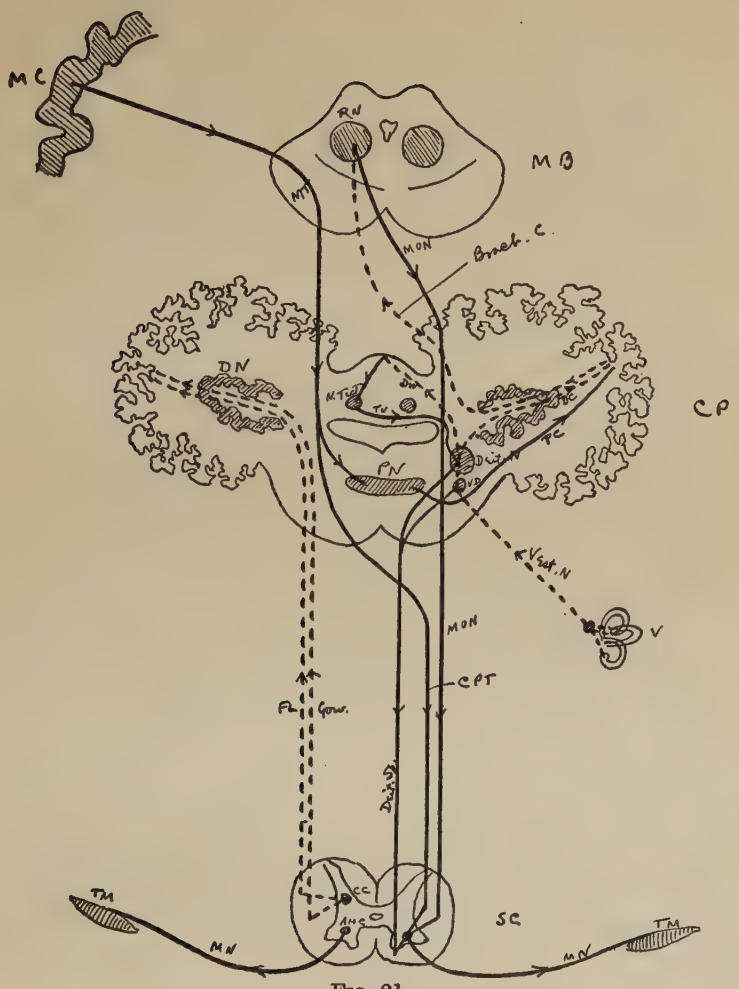

FIG. 21

TRACTS CONNECTING CEREBELLUM WITH SPINAL CORD, MID-BRAIN AND CEREBRUM, SHOWING PATHWAYS INVOLVED IN REACTION MOVEMENTS

MC—Motor cortex of cerebrum
MB—Mid-brain
CP—Cerebellum and pons
SC—Spinal cord
V—Vestibule
Vest.N—Vestibular nerve
RN—Red nucleus
DN—Dentate nucleus of cerebellum
Deit.N—Deiters' nucleus
VD—Ventro-caudal accessory Deiters' nucleus
NTect—Nucleus tecti
PN—Pontine nuclei

CC—Clarke's column of cells in posterior horn of spinal cord
AHC—Anterior horn cell
TM—Trunk muscles and muscles of extremities
MN—Motor nerves of muscles of trunk and extremities
FL—Flechsig's tract
Gow—Gowers' tract
DC—Tract from Deiters' nucleus to cortex of cerebellar hemisphere
DW—Tract from Deiters' nucleus to cerebellar worm
Brach.C.—Brachium conjunctivum
MT—Motor tract
Mon—Monakow's tract
TU—Tractus uncinatus
PC—Tract from pontine nuclei to cerebellar cortex
CPT—Crossed pyramidal tract. (Direct pyramidal tract is not indicated, for sake of clearness)
Deit.Sp.—Deiterso-spinal tract

cortex of each cerebellar hemisphere is connected with the anterior horn cells of the same side of the cord.

The following is the pathway for reaction movements of the body; from the vestibule through the vestibular nerve to Deiters' and the ventro-caudal nucleus. From here to the cerebellar worm *DW*. Then to the nucleus tecti *N Tect.* From here they pass through the tractus uncinatus *TV,* back to Deiters' nucleus, from which they pass through the Deiterso-spinal tract *Deit Sp,* to the anterior horn cells of the cord.

In addition to the centripetal impulses from the vestibule, sensory impulses reach the cerebellum through two tracts, **Flechsig's tract** or the direct cerebellar tract *Fl,* and **Gowers' tract** or the antero-lateral ascending cerebellar tract *Gow.* These tracts arise from the cells of **Clarke's** and **Stilling's columns,** at the base of the posterior horn of the spinal cord, around which the posterior nerve root fibres terminate. The fibres of these two tracts pass up through the lateral columns of the cord, and reach the cerebellum through its inferior peduncle. They pass through the central nuclei of the cerebellum to the cerebellar cortex.

The fibres of the motor tract, in passing down

through the pons, give off collaterals to the pontine nuclei. From here, fibres cross through the middle cerebellar peduncle to the cerebellar cortex of the opposite side.

In the cerebellar cortex motor impulses from the cerebrum meet impulses from the vestibule, and sensory impulses from the muscles, joints, etc., which pass up through Flechsig's and Gowers' tracts.

Following up the pathways of the impulses in the cerebellar cortex somewhat more in detail, we learn the following facts: Impulses pass from the vestibule to the dentate nucleus of the cerebellum. From here cells reach the dendritic processes of the cells in the granular layer of the cerebellar cortex (Fig. 22). The cells of the granular layer send axis-cylinder processes into the molecular layer, where they divide into two branches,

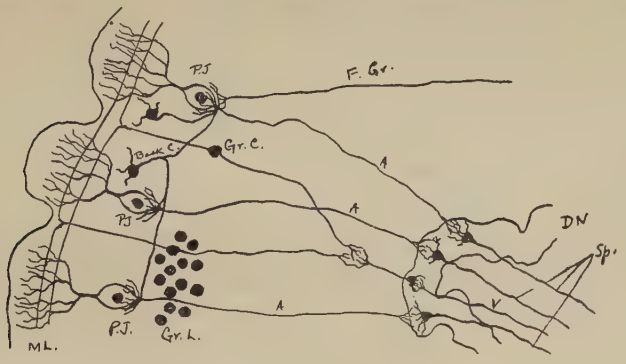

FIG. 22

PATHWAYS OF NERVE IMPULSES TO AND FROM CEREBELLAR CORTEX

ML—Molecular layer of cerebellar cortex
PJ—Purkinje's cells
Gr.L.—Granular layer
DN—Dentate nucleus
Bask.C—Basket cell
GrC—Cell in granular layer
FGr—"Fibre grimpante," passing from motor cortex of cerebrum to Purkinje cell
A—Axons of Purkinje cells
Sp—Tract to spinal cord
V—Tract from vestibular nuclei

running at right angles to the dendritic processes of the Purkinje cells, to which they transmit the impulses. In the molecular layer there are so-called basket cells, which terminate about the bodies of the Purkinje cells, in basket-like ramifications. The cells in the granular layer connect the rows of the Purkinje cells in the frontal plane, while the basket cells connect them in the sagittal plane. In this way each impulse from the vestibule is spread to the Purkinje cells over the entire cerebellar cortex. The impulses from the cerebral cortex, on the other hand, are individualized, each fibre surrounding one Purkinje cell, by means of the so-called "fibres grimpantes."

From the Purkinje cells the axis-cylinder processes pass down to the dentate nucleus. From here cells send axis-cylinder processes down to the spinal cord, in the tracts already mentioned (*Barany*).

CHAPTER II

PHYSIOLOGY

In the physiology of the vestibular structures and the semicircular canals, particularly of the latter, we have advanced much further than we have in the physiology of the cochlea, for the reason that animal experiments by a host of independent observers have led to many well-established conclusions. The function of the vestibular structures and the semicircular canals is separate and distinct from that of the cochlea. Although there is some ground for the belief that in the dog destruction of the cochlea is not followed by absolute deafness, yet in man there have been many instances where complete deafness was associated with a normally excitable static labyrinth. The cochlea is concerned only with hearing, while the utricle, saccule and canals (considered as a single organ) help to establish our body balance. These are therefore called collectively the static labyrinth, differentiating them thus from the auditory labyrinth (the cochlea). According to *Ewald,* the static labyrinth exercises a tonic influence over all the striated muscles of the body. It has been found in animals that an abnormal atony of the limbs develops after removal of the labyrinth, and that a general atrophy of the muscles ensues after plugging the semicircular canals. This theory has been supported by the later examinations of *Asch, Dreyfuss, Willgerodt, Bethe* and *Frölich,* who observed in lower animals a falling off of the motor force, with a simul-

taneous increase of the reflexes (*Politzer*). The view that any part of the so-called static labyrinth contributes to our perception or orientation of sounds has been abandoned.

The static labyrinth, however, is not the only source of impulses which help to maintain our equilibrium. *Flourens, Goltz, Breuer, Ewald* and many others have shown that, while the destruction of the static labyrinths is followed by marked loss of balance, this loss is only temporary.

The other sources of impulses for the preservation of our equilibrium perform the function of the static labyrinths vicariously, or, what is far more probable, the centres in which all these impulses are gathered accommodate themselves to the loss of the normal influence of the static labyrinths, and a compensation results. *Ewald* demonstrated that pigeons, both of whose labyrinths were destroyed several months before, behaved in a perfectly normal manner as regards their equilibrium, when subjected to certain tests, and he concluded that the destruction of both labyrinths entailed ultimately a complete loss of vertigo; i. e., animals with destroyed labyrinths could no longer become dizzy. Moreover, deaf-mutes whose labyrinths were destroyed by disease in early childhood, are able to maintain their equilibrium almost as readily as normal individuals.

Some believe that the static labyrinth in man is a rudimentary organ, but studies in comparative anatomy give no ground for such a conclusion. Moreover, if it were true, as some suppose, that this organ is normally quiescent and rarely gives rise to impulses, its sudden destruction would not cause the marked disturbances

to be observed in animals, nor would its destruction by disease in man be followed by the severe symptoms to be observed clinically. And if we do not see these severe symptoms frequently, it must be remembered that the destruction of the labyrinth by disease is, as a rule, a slow process, so that the centres gradually accommodate themselves to the loss of the normal impulses. It is true, however, that the more rapid the spread of a pathological process through the labyrinth the more severe are the symptoms.

Just how the impulses from the static labyrinths share with those from other sources in maintaining our equilibrium is a difficult matter to determine. Without doubt, however, all of these centripetal impulses combine harmoniously and, partly reflexly, at all events automatically, establish our balance without our realization of the processes involved. Every "disharmony" in these impulses which serve to maintain our equilibrium causes, if sufficiently pronounced, a marked disturbance in balance. If the discordant factor be but temporary, this disturbance will last until the normal state is re-established. If, however, the disturbing element be due to a permanent condition, such as the destruction of one or both static labyrinths, then the loss of body balance will continue until such time as the function of the destroyed organ or organs is performed vicariously, or the centres accommodate themselves to the absence of the normal impulses. The static labyrinth is probably always active, in the sense that it constantly sends to the centres impulses acquainting us with our position in space, and also with the changes in our position, i. e., motion in a straight line and rotation.

With regard to the physiology of the component parts of the static labyrinth, *Breuer* and *Mach* are of the opinion that the function of the maculæ (the end-organs of the saccule and utricle) is solely to enable us to orient the body in space and to perceive motion in a straight line. *Wittmaack* believes that the most important, if not the exclusive function of the vestibular apparatus is the development of impulses which acquaint us with the position of our bodies in space, and so help, with other impulses from the eyes, muscles, joints, etc., to establish our equilibrium. There is still, however, considerable uncertainty as to the manner in which the maculæ are stimulated. *Breuer* states that in mammals the macule of the utricle lies at right angles to that of the saccule, and he believes that the otolith membrane of the macula utriculi glides in a horizontal direction from without and behind, inwards and forwards, while that of the macula sacculi glides more or less vertically, from above and behind downwards and forwards.

Each macule contains a groove, and these grooves are at right angles to each other. In this way he assumes that, in accordance with the position of the head, the otolith membranes are acted upon by gravity and glide in their respective grooves. He was led to this gliding hypothesis by the peculiar anatomical construction of the maculæ in fishes. Here the nerve endings are distributed in a long groove in which the otolith membrane lies. In *Breuer's* opinion the otolith membrane can move only in the long axis of the groove and not at right angles to it. It has been shown, however, that these grooves frequently present irregulari-

ties which would make gliding of the otolith membrane, such as *Breuer* assumes, impossible. According to *Barany,* the question as to whether the otolith membrane does or does not glide is still unsettled. Moreover, he believes it unnecessary to assume such gliding, since all that he considers essential to stimulate the end-organ is that the otolith membranes because of their weight should be acted upon by gravity, in accordance with the position of the head, and so cause tension on or relaxation of the hairs of the hair-cells.

It has not been possible, up to the present, to stimulate the maculæ alone experimentally. It is evident that rotating the body must have some effect upon the maculæ as well as upon the cristæ, although the semicircular canals are, from their construction, better adapted to be aroused by turning than are the vestibular structures. If the axis of rotation passes through the otolith membrane itself, a torsion of the latter must result, with tension of the hairs on one side of the macula and relaxation of the hairs on the other side. Whether under these circumstances the tension and relaxation neutralize each other completely, so that no impulse results cannot be stated positively. Such a condition, however, is not probable. Moreover, this form of rotation must be very rare. The usual rotation occurs about an axis which does not pass through the otolith membrane, and here there is combined with the rotation a degree of forward motion which in all probability stimulates the maculæ. Then, too, the experiments of *Wittmaack,* who found the otolith membranes torn from the hair-cells after rapid rotation (2000 times per minute), show that centrifugal force probably

plays an important rôle in stimulating these end-organs.

In animals *Breuer, Lee, Kubo* and others have shown that certain reflex movements of the eyes, caused by movements of the head in space, are dependent upon the otolith organs. These movements of the eyes change with the various positions of the head. *Barany* states that these reflexes do not occur in normal man. In the blind, however, *Breuer* found eye movements analogous to the reflexes which occur in animals.

Barany ascribes sea-sickness, particularly that due to the pitching of a vessel, to stimulation of the otolith organs. The peculiar discomfort caused by rapid ascent or descent in elevators, "roller coasters" or gravitation roads he also believes to be due to the influence of the same end-organs. For he proved upon himself, while sea-sick, that by changing the position of his head forward or backward through an arc of 90°, so that he lay prone upon his face or upon his back, he felt no ill effects from the pitching of the boat, but as soon as he raised his head up from the horizontal position, even while lying on his back, he at once experienced nausea and was compelled to vomit. The same forward or backward movement of the head abolished the discomfort due to rapid ascent or descent in elevators, "roller coasters" or gravitation roads. He reasons that these phenomena can be caused only by an organ contained within the head, and he concludes that only the otolith organs come into consideration.

The foundation upon which the physiology of the semicircular canals is built we owe to *Flourens*. This observer noted that in pigeons a lateral movement of

the head with rotation of the body around its vertical axis occurred when the horizontal semicircular canal was cut through, that a movement of the head backwards and forwards, with a tendency of the body to fall backwards, occurred when the posterior canal was destroyed, and that destruction of the superior canal caused the animal to fall forwards. Despite the fact that *Flourens* observed these phenomena, he did not realize that he was dealing with an organ whose function it was to help maintain our equilibrium, but considered the semicircular canals a special apparatus to regulate the direction of our movements. His observations were confirmed by *Brown-Sequard, Vulpian, Goltz, Loewenberg, Cyon* and many other investigators. There was, however, no unanimity of opinion regarding the cause of the phenomena. Some experimenters failed to see in the severing of the semicircular canal an adequate cause for the reactions, and ascribed them to a variety of other causes, such as loss of blood, injury to the cerebellum, etc. Through the work of other investigators, however, and particularly through that of *Breuer,* the relation between the phenomena observed and the severed canal was finally established.

Goltz was the first investigator to grasp the true significance of the function of the semicircular canals. According to his view the semicircular canals are a special peripheral sense organ through which we become aware of the position of the head. The canals are, he continues, a sense organ for the establishment of the balance of the head and, through this, of the entire body. He believed that a stimulus was aroused

by the pressure of a column of endolymph upon the walls of the ampulla, and that this stimulus varied with the height of the column of endolymph which changed in different positions of the head.

In the vestibular structures, the otolith membranes, because of their weight, cause a degree of pressure on the hairs of the hair cells which develops a continuous impulse, a rest-tone (Ruhetonus) as it were. Whether such an impulse exists in the semicircular canals has not yet been determined. While the difference in specific gravity between the cupula and the surrounding endolymph is not sufficient to warrant such a conclusion, yet it is probable that a rest-tone does exist in the cristæ, even though it may be secondary in physiological importance to that of the maculæ. Besides, it may be aroused in some way not dependent upon the weight of the cupula, but through some influence upon this structure which causes it to undergo a constant movement either molecular or en masse.

The chief function of the semicircular canals is unquestionably to respond to rotation of the body and acquaint us with the changes in the speed and direction of such rotation, so as to help maintain our equilibrium.

It is the generally accepted opinion that the end-organ in each semicircular canal is its crista, and that the process which stimulates this is a movement of the endolymph, which, when it reaches the cupula, displaces the latter from the position it occupies when the body is at rest. This movement of the cupula must be exceedingly small, according to *Barany* .01 mm. or less, for any considerable movement, such as that caused by

cutting through the canal, would tear the cupula from the hair cells.

As to the manner in which rotation of the head arouses a stimulus in the canals, let us consider for a moment the left horizontal semicircular canal during rotation of the body (head) to the left. At the beginning of rotation the endolymph, because of its inertia, remains stationary while the canal moves. This is equivalent to a movement of the endolymph in a direction opposite to that of the rotation of the body, and causes a displacement of the cupula toward the utricle, with the corresponding reaction. If the rotation is continued at a uniform speed, the endolymph gradually acquires a rate of speed equal to that of the canal, and the cupula, partly for this reason and partly through the elasticity of the hairs which project into it, is returned to its usual position. If the rotation cease suddenly, then the endolymph, because of its momentum, continues to move in the direction of the turning (left) while the canal is still, and the cupula is displaced away from the utricle. Here again, because of the elasticity of the hairs and the cessation of movement in the endolymph, the cupula regains its usual position. The cupula itself shares with the endolymph the inertia at the beginning of the rotation, and the momentum when the rotation ceases.

These phenomena occur irrespective of the speed or duration of the turning, but their intensity varies with these, and the central reactions to impulses of varying degrees of strength, differ decidedly. If the rotation is slow or if it comprise but a small arc of a circle, the apparent reaction when rotation has ceased is nil. On

the other hand, if the rotation is rapid and through a number of turns, there is a manifest reaction when it has ceased. In our opinion, however, there is no ground for the belief that there exists an essential difference between the impulse aroused by the former and that aroused by the latter. How, then, can we explain the fact that rotation which is slow or lasts but a short time calls forth no apparent reaction? We believe that the static labyrinth is always active and responds to the slightest rotation, for it is apparent that in reacting to these slight turns this organ performs its usual function. The centres of the vestibular nerves, however, have a degree of tolerance, which varies not only in different individuals, but at different times in the same individual. It is established by a series of checks and balances brought about by the influence of all the centripetal impulses whose function it is to preserve our equilibrium. So long then as the strength of an impulse aroused in the labyrinth does not exceed this tolerance limit, the centres are able to accommodate themselves to it, and no apparent reaction occurs. As soon, however, as such an impulse exceeds this tolerance limit the checks and balances are no longer able to control, and a manifest reaction supervenes.

Breuer, who in his experiments took no account of nystagmus, believed that the sensation of turning when rotation had ceased was dependent upon and synchronous with the displacement of the cupula. Although we accept the former hypothesis, the latter does not agree with the facts. According to *Barany,* if we rotate a number of individuals, the sensation of turning after rotation has ceased varies in duration in different in-

stances despite the fact that the manner of rotating is the same in each. If we carefully observe the nystagmus after rotation, we will frequently find that of two individuals who have nystagmus lasting equally long, one will have the sensation of turning synchronous with his nystagmus, while the other feels little or no sensation of turning at all. Nystagmus is a far better indication of the stimulation of the cristæ than is the turning sensation, for the former can be observed objectively. Thus the fact that in two cases the nystagmus lasts equally long, while the duration of the turning sensation is unequal, controverts *Breuer's* hypothesis. Although we accept as a fact that so long as the cupula is displaced there is a change in the normal centripetal impulse, we cannot believe that the condition of the end-organ is alone responsible for the duration of the reaction. This is true as well for nystagmus as for the turning sensation. If we rotate an individual he will have a far stronger reaction if we cease after ten turns, than if we cease after thirty turns. This fact alone demonstrates that the centres play an important part in controlling the reactions to these peripheral stimuli.

In an exceedingly interesting recent communication on this subject *Shambaugh* takes the position that the duration of nystagmus is coextensive with the peripheral stimulation, and that the latter lasts as long as there is an endolymph current.

Before entering into a discussion of the effect of endolymph movements upon the crista, it is necessary to call attention to some anatomical peculiarities of the latter, which have a bearing upon the physiology of this organ. The crista is a comb-like hill of cells

placed at right angles to the course of the canal. It is composed of hair-cells held in place by supporting cells. While in many animals the hair-cells are absent in a zone surrounding the apex of the crista, in man they cover the latter from its base to its apex. Each hair-cell has a number of cilia, which run free a short distance out of the cell, and then pass into canals in the overlying gelatinous cupula. These canals are longer than the contained hairs. *Breuer* and *Kolmer* state that the cupula is apparently secreted by the supporting cells of the crista and is constantly wearing off at its upper part. For this reason the canals are longer than the hairs.

According to *Cajal* the nerves of the crista are not uniformly distributed. The cells of the upper part are far more richly supplied with nerve filaments than are the cells of the remaining portions. This probably indicates a greater functional activity of the upper cells. Whether the nerves actually reach into the cells or merely surround them is irrelevant, so far as the physiology of the organ is concerned. For it is of no import whether the stimulus reaches the nerve endings directly or is translated to them from the cells.

With regard to the process through which stimulation of the crista by endolymph movements is transformed into nerve impulses, *Breuer* has developed an interesting theory, which, while it does not satisfy all objections to it, yet offers a working hypothesis and goes far toward clarifying our views concerning the physiology of this organ. *Breuer* assumes that a movement of the endolymph which displaces the cupula toward the utricle causes tension on the hairs of the

canal side of the crista and relaxes those of the utricular side. He assumes further that only tension on the hairs develops a stimulus. If a rest tone, such as has been described above, exists in the crista, then it follows that relaxation of the hairs, by disturbing this rest tone, would also produce a stimulus. In man, the hair-cells cover the entire crista, and the hairs of the cells situated at the apex would be made tense whether the cupula were moved toward or away from the utricle.

It is necessary, therefore, to assume that the hairs of the apical cells react in such a way that, irrespective of whether the cupula is moved toward the utricle or away from it, tension arouses the central equivalent of a movement in only one of these directions. Only the corresponding central relations are necessary to bring about such a state of affairs. In other words, just as any stimulus to the retina arouses an impulse which is always translated centrally into the sensation of light, so tension on these hairs at the apex of the crista, no matter how such tension is caused, arouses the central equivalent of a movement of the endolymph in only one direction. With this hypothesis it is easy to explain the results of the classical experiments of *Ewald*. The latter proved that while the mechanical irritation remained the same, the intensity of the reaction varied, depending upon the direction of the movement of the endolymph, and therefore of the cupula. Thus he showed that in the horizontal canal a movement of the endolymph toward the ampulla causes a reaction about twice as strong as a movement toward the small end. In the vertical canals (the superior and posterior) the reverse of this is true, i. e., the movement of

the endolymph toward the ampulla causes the weaker reaction.

Let us examine these processes of stimulation for a moment in the light of the foregoing hypothesis. Given a movement of the endolymph in the horizontal semicircular canal toward the ampulla, there would follow a displacement of the cupula toward the utricle. Hereupon the hairs of the apex and of the canal side would be made tense, while those of the utricular side would be relaxed. Let us now assume that in the horizontal semicircular canal, tension on the apical hairs of the crista arouses the central equivalent of a movement of the cupula **only toward the utricle.** Then the central reaction to a movement of the cupula toward the utricle would be the resultant of a stimulus from the apex cells, plus that from the canal-side cells, minus that from the utricular-side cells, provided, of course, that relaxation of the hairs is capable of producing a negative reaction. If we suppose the displacement of the cupula to be away from the utricle, then the central reaction would be the resultant of the stimulus from the utricular-side cells, minus that from the apex cells as well as that from the canal-side cells. If the peculiar distribution of the nerves in the crista, as shown by *Cajal,* has any physiological significance, i. e., if the apex cells exercise proportionately a greater functional activity than the cells of the rest of the crista, then we have in this an additional cause for the fact that in the horizontal semicircular canal a movement of the cupula toward the utricle is followed by a far stronger reaction than a movement in the opposite direction. It must be apparent that in any group of hairs the impulse

aroused by tension (plus impulse) exceeds in strength that aroused by relaxation (minus impulse).

For the apex cells of the cristæ in the vertical canals, we must assume such central relations, that the impulse aroused by tension on their cilia is translated centrally into a movement of the cupula only away from the utricle.

As has been stated above, every "disharmony" in the impulses which serve to maintain our equilibrium causes, if sufficiently pronounced, a marked disturbance in balance. The character of this disturbance varies with the physiological peculiarities of the end-organ in which the "disharmonic" impulse arises. The function of the static labyrinth is to acquaint us with our position in space and with the changes in that position, i. e., acceleration or retardation of motion in a straight line and rotation. Under ordinary circumstances there are constantly aroused with these perceptions of our position and its changes, reflex stimuli, whose purpose it is to cause the eye muscles and the body muscles to accommodate themselves to the conditions that call forth these perceptions. If, however, an impulse is aroused in the labyrinth so strong that the central checks and balances can no longer control, then there ensues a disturbance of the equilibrium whose characteristics depend upon the functional activities of this organ. Thus there occur disturbances in our perception of position, sensation of turning, along with disturbances of the eye muscles (nystagmus), and disturbances in the body muscles (reaction movements). Finally, if the impulse is very strong, radiation from the central vestibular nuclei, in the floor of the fourth ventricle, to the vagus

nuclei occurs and nausea and vomiting supervene. Such a powerful impulse may be aroused in the semicircular canals in various ways; mechanically by rotation or by air pressure if there be a fistula; calorically by hot or cold water or other fluids; chemically by the application of substances directly to the exposed membranous canal; and electrically by the application of the galvanic current.

Of the reactions which follow a strong impulse aroused in the semicircular canals, the most important by far, from a clinical standpoint, is **nystagmus.** This may be defined as a rhythmic to-and-fro motion of the eyes, consisting of two components, a slow movement in one direction, which is followed at once by a rapid movement in the opposite direction. Although it has been proven (*Ewald, Barany* and others) that the slow component is the vestibular reflex, the reaction has been named according to the direction of the rapid component. Thus we speak of nystagmus to the right when the rapid component is directed to the right.

Ewald has shown that nystagmus always occurs in the plane of that semicircular canal in which the impulse calling forth the nystagmus arises. He proved further that the slow component always takes the same direction as the endolymph movement, or that the rapid component takes a direction opposite to that of the endolymph movement.

If a stimulus is aroused in the labyrinth during a state of unconsciousness, only the vestibular reflex (slow component) appears, while the voluntary reaction (rapid component) is abolished. If we perform a caloric test of a labyrinth upon a patient during narcosis

the eyes will deviate to the side, toward which the slow component would be directed if there were a nystagmus. From the fact that the slow component is the vestibular reflex, while the rapid component is influenced by the will, it is easy to understand why the rapidity of nystagmus is influenced by the direction in which the eyes are fixed. The greater the effort to fix the eyes in the direction of the rapid component, the more rapidly the eyes are drawn back after the conclusion of the slow component. *Barany* observed that, when the eyes were directed toward the side of the rapid component, the slow movements were smaller but more frequent.

The rapidity of the nystagmus, however, depends primarily upon the rapidity of the rapid component, and not, as some hold, upon the resistance to the slow component. If the resistance to the slow component increased as the eyes were turned toward the side of the rapid component, it would be impossible to explain (1) that an artificially aroused nystagmus becomes apparent first when the eyes are turned to the side of the rapid component; (2) that after such a nystagmus is aroused it may frequently be abolished by fixing the eyes in the direction of the slow component; (3) that spontaneous nystagmus may be abolished by directing the eyes toward the side of the slow component. These facts seem to us to prove that the resistance to the vestibular reflex (slow component) increases as the eyes are turned away from the side of the rapid component, and that this resistance is greatest when the eyes are directed to the side of the slow component.

In a way, this resistance gives us a gauge of the intensity of the nystagmus. If the latter becomes appar-

ent only when the eyes are turned to the side toward which the rapid component is directed, we speak of a nystagmus of the first degree. If the nystagmus is evident when the eyes are directed forward as well, we speak of a nystagmus of the second degree, and if the nystagmus is apparent even when the eyes are directed toward the side of the slow component, we speak of a nystagmus of the third degree. When nystagmus is aroused artificially it becomes apparent first, as has been mentioned, when the eyes are turned to the side toward which the rapid component is directed. In the extreme lateral positions of the eyes there are frequently to be observed nystagmic movements which are physiological. These are directed to the side toward which the eyes are turned, are usually horizontal, but sometimes rotatory, and are as a rule of short duration.

Since the rhythmic movements of the eyes in vestibular nystagmus result exclusively from the reflex stimuli aroused by vestibular irritation, they are most clear when least influenced by voluntary, restraining movements of the eyes. Thus they are most marked when the eyes are closed or in individuals who are blind. Fixing the vision may influence vestibular nystagmus not only by causing accommodation but also by arousing optical nystagmus. The former by the associated convergence of the eyes causes one of them to turn toward the side of the rapid component, while the other takes the opposite direction. The latter (optical nystagmus) is usually caused by an attempt to fix the vision upon constantly changing objects, which have the same relative position to the body and actually or apparently move by.

The best example of this form of nystagmus is seen in the eyes of an individual who looks out of the window of a rapidly moving car. In the presence of a vestibular nystagmus, an optical nystagmus is aroused by attempting to fix the vision upon an object situated to one side or the other, which apparently moves by. This results in a to-and-fro motion of the eyes which parallels the vestibular nystagmus. It must be apparent, then, that if this optical nystagmus is directed to the same side as the vestibular nystagmus, they accentuate each other, while if the optical nystagmus is directed opposite to the vestibular nystagmus they oppose each other.

A vestibular nystagmus to the right begins with a slow movement toward the left, which is followed at once by a rapid movement to the right. The observations of *Högyes,* which were substantiated by *Bartels,* show that if all the ocular muscles, with the exception of either the rectus internus or externus, be dissected free from the bulb, the remaining muscle alone is capable of executing nystagmic movements in both directions. *Bartels* separated the muscles from the eye ball and arranged an apparatus in such a way that each muscle wrote for itself its contractions and relaxations. Thus he showed that the rectus internus or externus of either eye is capable of executing nystagmic movements both to the right and to the left.

If we consider alone the activity of the rectus externus of the right eye during a nystagmus to the right, we find that the slow component to the left can occur only through a slow relaxation of this muscle, while the rapid component must result from a rapid contrac-

tion. For the rectus internus, the reverse of this is true, i. e., the slow component to the left results from a slow contraction of this muscle, while the rapid component to the right is due to a rapid relaxation. *Bartels* has proven that these muscular phenomena actually occur. The muscular activities of the left eye, during a nystagmus, are the reverse of those that occur in the right. Thus while during the slow movement to the left, the right rectus externus slowly relaxes, the left rectus externus slowly contracts. The eye ball in describing the slow component, starts from its position of rest, so that if this movement to the left is executed in the right eye by the rectus externus alone, there must be an abolition of the normal rest-tone of this muscle which results in an "active relaxation."

An endolymph movement in the right horizontal semicircular canal which displaces the cupula toward the utricle has, as a result, a nystagmus directed toward the right. In order to produce the slow component of this nystagmus the following muscular phenomena must occur:

Right Eye { 1. Slow relaxation of the rectus externus.
2. Slow contraction of the rectus internus.

Left Eye { 1. Slow contraction of the rectus externus.
2. Slow relaxation of the rectus internus.

According to *Barany,* the simultaneous contraction of one set of muscles with the relaxation of the other set can be considered of peripheral origin, provided we accept the presence of a rest-tone in the cristæ of the canals. He believes, further, that from these muscular phenomena he can deduce relations between definite

groups of hair cells and the central nuclei of individual eye muscles. Whether this be true or not, and whether the muscular mechanism of nystagmus is controlled entirely by conditions in the peripheral organ (tension or relaxation of the hairs) or is solely under the dominion of the centres, one fact stands out clearly, viz., either labyrinth is in relation with the central nuclei of the muscles of both eyes, and a powerful impulse aroused in one labyrinth, even after the complete destruction of the opposite labyrinth, is capable of causing both eyes to execute nystagmic movements in all planes, and directed either to the sound side or to the other.

In addition to nystagmus, there are other phenomena which result from powerful impulses aroused in the semicircular canals. These reactions are partly subjective and partly objective. The objective phenomena **(reaction movements)** are so closely allied to the cerebellar tests of *Barany,* that their further description will be deferred until the discussion of that subject. The subjective phenomena consist of sensations, on the part of the individual in whom the semicircular canals are stimulated, of being turned himself and of an apparent turning of the objects in his environment.

These sensations bear a striking analogy to the nystagmus, but are less uniform in their behavior than the latter, and are therefore far less valuable as tests of labyrinthine function. The sensation of turning varies considerably in different individuals who have been subjected to the same tests. In some it is quite marked, while in others it is almost totally absent. It is felt most clearly when the eyes are closed. As a rule the

apparent turning of the body is in the direction of the rapid component, and like the nystagmus is influenced by the direction in which the eyes are fixed. If the eyes are fixed in the direction of the rapid component the turning sensation is increased. Fixing the eyes in the opposite direction has, of course, the opposite effect. The apparent turning or passing by of objects in the environment of an individual who has a vestibular nystagmus also varies. As a rule the objects seem to pass in the direction of the rapid component. Sometimes, however, they seem to oscillate, tip over or even move in the direction of the slow component. The manner in which objects seem to move is evidently the result of the retinal impression, as a rule during the progress of the slow component.

Van Rossem has carefully worked out the relation between the sensation of being turned, the reaction time, etc., and the speed of rotation, but as yet these facts have not attained any clinical significance.

The disturbances in our sensation of position are doubtless due to some extent, at least, to impulses from the otolith organs as well as from the cristæ, particularly if the labyrinth is stimulated by rotation. Here, of course, the otolith membranes must be affected by centrifugal force.

During a horizontal nystagmus, nausea, vomiting, disturbances in the heart's action and in respiration rarely occur, but they are not at all uncommon in the presence of either a vertical or a rotatory nystagmus. *Barany* has shown that sea-sickness, with its attendant symptoms, is largely due to the influence of the otolith organs. Moreover neither *Neumann* nor *Ruttin* ob-

served nystagmus during sea-sickness, so that the semicircular canals probably play little or no part in this. These facts seem to indicate a closer central relationship between the vertical canals and the otolith organs, than between the latter and the horizontal canals.

All the centripetal impulses, whose purpose it is to maintain our equilibrium, are gathered together in the cerebellum, so that the latter may be considered, in a sense, the centre for the static labyrinth. There probably is no single, definite (static sense) centre upon which all of the centripetal impulses exert their influences. The cerebrum must play a very subordinate rôle in this static sense, however, for it has been entirely removed in dogs without causing any loss of equilibrium. On the other hand, removal of even half the cerebellum results in very decided loss of balance.

The cerebellum may be considered as a sensory-motor subcortical central organ. It receives centripetal impulses from the static labyrinth, sensory impulses from the muscles and joints and visual impulses from the eyes, and it sends out motor impulses to the muscles of the head, trunk and extremities, the purpose of which is to maintain the equilibrium of the body.

All of the motor impulses, however, which have for their purpose the maintenance of the body equilibrium, do not originate in the cerebellar cortex. Some of the movements are more or less under the dominion of the will, and these originate in the motor area of the cerebral cortex. The impulses which arouse these movements, however, pass through the cerebellar cortex on their way down to the spinal cord. In the cerebellar cortex they meet the centripetal impulses from the static

labyrinths, the muscles, the joints and the eyes, and are there acted upon in such a way as to become effective in maintaining the equilibrium of the body. According to *Lewandowsky,* the function of keeping the body in a condition of equilibrium may be described as that combined action of the muscles of the extremities, trunk, head and eyes which results in the holding of the body in an upright symmetrical position, in the possibility of moving about in certain planes and directions, and in the compensation for passive rotation of the head and body.

The connections between the static labyrinth and the cerebellum, between the joints and muscles and the cerebellum, and between the cerebral cortex and the cerebellum have been carefully worked out.

Impulses from the vestibule and semicircular canals pass through the vestibular nerve to Deiters' nucleus. From Deiters' nucleus a pathway leads to the corpus dentatum of the cerebellum, and from here to the cortex of the worm and cerebellar hemispheres of both sides.

Sensory impulses from the muscles and joints pass into the spinal cord through the posterior nerve roots, where the fibres terminate about the cells in Clarke's and Stilling's columns at the base of the posterior horns. From these cells fibres pass up through the lateral tracts of the cord in Flechsig's and Gower's columns. These fibres reach the central nuclei of the cerebellum through the inferior cerebellar peduncles. From the central nuclei fibres reach the cerebellar cortex.

From the motor cortex of the cerebrum impulses

pass down through the internal capsule and the crura cerebri to the pons. Here collaterals are given off, which terminate in the pontine gray matter. From the latter, fibres pass across through the middle cerebellar peduncle to the cerebellar cortex of the opposite side. From the cerebellar cortex, according to *Lewandowsky*, motor impulses pass down in two ways: 1. From the cortex of the cerebellar hemispheres to the dentate nucleus. From here through the brachium conjunctivum to the red nucleus of the tegmentum of the opposite side. From here fibres pass down through Monakow's bundle, across to the opposite lateral tract of the spinal cord, where they terminate about the anterior horn cells. 2. From the cortex of the cerebellar worm to the nucleus tecti. From here through the tractus uncinatus to Deiters' nucleus and the ventro-caudal Deiters' of the opposite side. From here, impulses pass down through the Deiterso-spinal tract and the posterior longitudinal bundle of the cord to the anterior horn cells.

Thus it is seen that the cerebellar hemispheres are connected with the anterior horn cells of the same side of the spinal cord.

Impulses reach Purkinje's cells in the cerebellar cortex, from the vestibular apparatus, muscles and joints, through the intermediation of the cells of the granular layer. The axis-cylinder processes of the granule cells are distributed to a large number of Purkinje cells. The Purkinje cells are further joined together by means of the basket cells. In this way each impulse from the static labyrinth is transmitted to a large number, or possibly even all of the Purkinje cells. On the other

hand, the impulses from the cerebrum reach Purkinje's cells through the so-called "fibres grimpantes," one of which goes to only one Purkinje cell. This connection is therefore strictly individualized. From Purkinje's cells the impulses pass down through the motor tracts to the anterior horn cells of the spinal cord.

The pathways concerned in the nystagmus reflex are as follows: from the static labyrinth impulses pass through the vestibular nerve to Bechterew's and the ventro-caudal Deiters' nucleus. From here impulses pass to the posterior longitudinal fasciculus of both sides, the fibres of which pass up through the pons and mid-brain, where they terminate about the nuclei of the third, fourth and sixth cranial nerves. Through these nerves impulses pass to the ocular muscles.

The pathways through which reaction movements of the body and the extremities are intermediated, are not so direct, since the cerebellar cortex is involved in this circuit. The pathways for the reaction movements of the extremities differ in part of their course from those for the reaction movements of the body. Impulses for the reaction movements of the extremities pass from the static labyrinth through the vestibular nerve, to Deiters' nucleus and the ventro-caudal accessory nucleus. From here they pass through the dentate nucleus to the cortex of the cerebellar hemispheres. From here they pass through the brachium conjunctivum to the red nucleus of the tegmentum of the opposite side. From here they pass through Monakow's tract to the lateral columns of the cord on the opposite side, where they terminate about the anterior horn cells.

Impulses for reaction movements of the body pass from the static labyrinth, through the vestibular nerve, to Deiters' nucleus. From here they pass to the cortex of the cerebellar worm. They then pass down to the nucleus tecti, from which fibres pass through the tractus uncinatus, to Deiters' nucleus and the ventro-caudal nucleus of the opposite side. From here they pass through the Deiterso-spinal tract to the anterior horn cells, which send the impulses to the muscles of the body.

The voluntary element of the reaction movements reaches the cerebellar cortex by way of collaterals which are given off from the motor tract as it passes through the pons. These collaterals terminate about cells in the pontine gray matter. These cells send neurons through the middle cerebellar peduncles to the cerebellar cortex.

PHYSIOLOGY OF THE COCHLEA

The cochlea is the end-organ which is concerned in the sensation of hearing. Just how the cochlea utilizes the sound waves, and transforms them into impulses which are perceived as sound is not definitely known. Our knowledge in this field is based entirely upon theoretical considerations.

All are agreed that the sound waves are taken up in some way by the hair cells of the organ of Corti, and transformed by them into nerve impulses, which pass through the fibres of the cochlear nerve to the cortex of the temporo-sphenoidal lobe of the brain, where they are recorded as sensations of sound; but here the

unanimity of opinion ends. There are innumerable theories as to how the sound waves are imparted to the hair-cells, and as to whether the analysis of sound occurs in the cochlea, or in the cortex of the brain.

The majority of physiologists and otologists seem to be of the opinion that the sound analysis occurs in the cochlea. In support of this theory are the following facts: the extreme complexity of the cochlea, with the many thousands of hair cells and nerve fibres; the occurrence of tone islands, i. e., the absence of hearing for certain notes when portions of the cochlea are diseased; the occurrence of diplacusis, i. e., the double hearing of a single tone analogous to double vision; and the experiments of *Wittmaack, Siebenmann* and *Yoshii,* who showed that certain definite portions of the cochlea degenerated in animals, as the result of exposing them for a long time to a continuous sound of a certain pitch.

One of the oldest, and perhaps the most widely accepted of the theories which postulate peripheral sound analysis, is the resonator theory of *Helmholtz. Helmholtz's* theory of hearing is as follows: The molecular air vibrations of sound, on reaching the membrana tympani, are transformed into molar or mass vibrations. The membrana tympani is so constructed that it vibrates equally well for all tones. That is, it is not attuned to any special tone which it will pick out and magnify. From the membrana tympani the vibrations are carried through the three ossicles to the foot-plate of the stapes, by which the impulse is imparted to the perilymph in the vestibule. Since the perilymph fluid is much more dense than air, it is necessary for the

sound vibrations to be increased in intensity, in order to produce adequate stimulation of the hair cells. In increasing the intensity the extent of the oscillations is diminished.

It is the function of the membrana tympani and the ossicular chain to transform oscillations of slight intensity and large range into oscillations of greater intensity and lesser range. This is brought about in three ways: first by the difference in size between the membrana tympani and the foot-plate of the stapes, the former being many times larger than the latter. The impulse which is imparted to the large surface of the membrana tympani is concentrated onto the smaller surface of the foot-plate of the stapes, and is consequently increased in intensity. Second, the handle of the hammer is half again as long as the vertical process of the incus. The shortening of this lever again increases the intensity of the force. Finally, the shape of the membrana tympani has a similar effect. The drum membrane is shaped like a funnel, at the apex of which is the end of the hammer handle. The sides of the funnel are curved, the convexity being directed outward. When an impulse is imparted to the convex membrane the latter has a tendency to flatten. The impulse is transmitted to the apex of the funnel,where the movement is diminished in extent and increased in force.

The foot-plate of the stapes transmits the impulse to the perilymph fluid in the vestibule. The impulse passes through the perilymph in the scala vestibuli to the apex of the cochlea, where it passes through the helicotrema to the scala tympani. It then passes down

through the scala tympani to the round window, where it sets into vibration the secondary tympanic membrane.

From the perilymph in the scala vestibuli and tympani the impulse is transmitted to the endolymph in the ductus cochlearis through Reissner's membrane and the basilar membrane.

If a note is struck on a piano, and there is in the vicinity another string of the same length as the piano string which was struck, this second string will vibrate in harmony with the first. Each tone has a string of a certain length which can be made to vibrate in harmony with it. The higher the pitch of the tone, the shorter is the string which is made to vibrate in harmony with it. Columns of air can be made to vibrate in harmony with a tone in the same way as strings. Cylinders, which contain such columns of air, are called resonators, and are used to intensify the volume of tones.

The membrana basilaris, although a membrane, can be considered a series of strings, as it consists of radiating fibres bound together by a soft, interfibrillary substance. The fibres pass from the bony spiral lamina to the ligamentum spirale. It is estimated by *Hasse* and *Retzius* that there are from 15,000 to 25,000 fibres in the basilar membrane. They are shortest at the beginning of the basal whorl and gradually become longer toward the apex of the cochlea. According to *Hensen* they are 0.041 mm. long at the base and 0.495 mm. long at the apex of the cochlea. In other words, they are twelve times as long at the apex as at the base.

Helmholtz considered that each one of the fibres can vibrate independently of all the rest. Each fibre vibrates in harmony with a certain tone. When the vibra-

tions causing a certain tone are carried to the perilymph, the fibre which is in harmony with that tone is set into vibration. The vibration is transmitted to the hair-cells overlying this fibre, and converted by the hair-cells into nerve impulses, which are carried by the fibres of the cochlear nerve to the brain. There are as many nerve fibres as there are hair-cells. Thus each nerve fibre always transmits the impulse aroused by the same tone.

In reality *Helmholtz* did not consider that one fibre vibrated with each tone, but that a group of fibres was set into vibration, the central fibre vibrating strongest, and the vibration gradually diminishing in the fibres on either side. The vibration of the central fibre is so much stronger than the others that the tone to which it corresponds predominates.

The fibres at the base of the cochlea, being the shortest, intermediate the highest tones, and those at the apex, being the longest, intermediate the lowest tones. The normal range of hearing is from 16 double vibrations to 40,000 double vibrations per second.

Ewald believed that the fibres of the membrana basilaris do not vibrate individually, but that the entire membrane vibrates as a whole. He stretched a rubber membrane, shaped about like the basilar membrane, on a wooden frame, and imparted the vibrations of tuning forks to it. By coating the rubber with oil, he was enabled to see the waves of vibration on the membrane. Each tone gave rise to standing waves of certain shape, which were constant. The higher tones gave rise to narrow waves, which were close together, and the lower tones to broader waves, which were further apart.

There is a certain "vibration picture" for each tone. *Ewald* believed that such vibration pictures occur on the basilar membrane, and that all the hair cells at the apices of the waves being stimulated, gave rise to the sensation of hearing for that tone.

Barth believes that sound waves are transmitted to the perilymph fluid, not as mass movements but as molecular movements. Whether the oscillations which are transmitted through the membrana tympani and ossicles are molecular or molar is doubtful, but sound waves are unquestionably transmitted to the perilymph directly through the bones of the skull in the form of molecular motion, although part of this motion is imparted to the membrana tympani and reaches the perilymph through the ossicular chain.

Barth believes that the acoustic nerve impulses are not produced by a movement of the hairs of the hair cells with the movement of the endolymph, but by the resistance offered by the hairs to the movement of the endolymph.

It is not probable that the vibration is transmitted directly to the hair-cells from the endolymph without intermediation, for there is no apparent difference between the individual hair-cells such as would make it probable that each cell would respond to a different tone.

Hasse, Siebenmann, Von Stein, Kishi, Hardesty and *Shambaugh* believe that it is not the basilar membrane but the membrana tectoria whose vibrations stimulate the hair-cells. *Shambaugh* found that in parts, the basilar membrane was so thick and rigid that it could not possibly vibrate.

At the beginning of the basal whorl he found it absent, although there was a perfectly formed Corti's organ present here. The vas spirale is attached to the lower surface of the basilar membrane. As this vessel is sometimes dilated and at other times contracted, it must change the resonating quality of the radiating fibres of the basilar membrane, from time to time, so that the same fibre is not always attuned to a tone of the same pitch.

In order to overcome these objections, *Shambaugh* has assumed the tectorial membrane to be the vibrating organ. The impulse is transmitted directly from the scala vestibuli through Reissner's membrane to the tectorial membrane. The tectorial membrane lies directly over the hair cells, the hairs being imbedded in the substance of the membrane. In this way movement of the tectorial membrane is imparted directly to the hairs of the hair cells. This theory brings the action of the end-organ in the cochlea into harmony with the action of the end-organs in the vestibule and semicircular canals. The otolith-membrane in the vestibule, the cupula in the ampullæ of the semicircular canals, and the tectorial membrane in the organ of Corti all overlie the hair-cells.

The hairs are embedded in these membranes, and movement in the membranes is imparted directly to the hairs. The tectorial membrane varies in size at different parts of the cochlea, being smallest at the beginning of the basal whorl and largest at the apex of the cochlea.

Rutherford considered that the entire basilar membrane vibrated with every tone, very much as the disc

of a telephone receiver vibrates. Each tone is carried to the brain by all of the nerve fibres of the cochlear nerve, and the sound analysis takes place in the brain. This is known as the telephone theory.

W. S. Bryant supports this theory of central analysis by a report of twenty-six ears collected from the literature in which functional tests were made, and, after death, complete histological examinations. In these cases there was no uniformity in the relationship between the tones that were not heard and the portions of the cochlea which were diseased.

In contradiction to these findings, however, are the results of experiments made on animals by *Wittmaack, Siebenmann* and *Yoshii.* They subjected guinea-pigs to the hearing of the same tone repeated over and over again, through a long period of time. The animal was then killed and the cochlea examined histologically. In these animals, where a tone of high pitch was employed, there were regularly found degenerative changes in

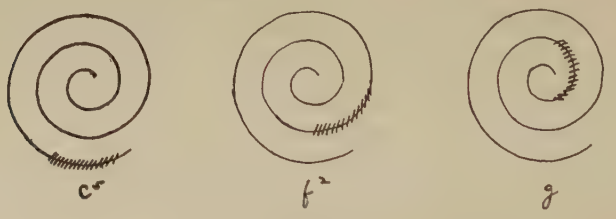

FIG. 23

DIAGRAMS ILLUSTRATING PORTIONS OF COCHLEA SHOWING DEGENERATIVE CHANGES IN SIEBENMANN'S EXPERIMENTS WITH GUINEA-PIGS, EXPOSED TO THE CONTINUOUS SOUND OF A TUNING FORK

C^5—Shaded portion indicates region showing degenerative changes when C^5 fork is used

f^2—Shaded portion indicates region showing degenerative changes when f^2 fork is used

g—Shaded portion indicates region showing degenerative changes when *g* fork is used

Corti's organ in the basal whorl of the cochlea. Where a low-pitched tone was used, the degenerative changes appeared in the upper whorls.

Siebenmann exposed a guinea-pig to the sound of a C^5 whistle for several hours each day during several months. On examining the cochlea he found degenerative changes in the organ of Corti in the basal whorl (Fig. 23). In a guinea-pig which was subjected to the tone of the F^2 whistle, degeneration was found in the middle whorl. In a guinea-pig subjected to the tone of a G pipe the changes were found in the upper whorl. That these degenerative changes were not accidental nor due to post-mortem changes was proven by the fact that the same changes were found repeatedly, in different guinea-pigs, when exposed to sound of the same pitch.

CHAPTER III

METHODS OF EXAMINATION

The functional examination of the static labyrinth may be divided into two parts:

1. The determination of the presence or absence of signs and symptoms which indicate a loss, a diminution or an increase in the normal impulses from the static labyrinth (decompensation symptoms).

2. The determination of the condition of the static labyrinth by observation of certain reflexes, which are elicited by physiological stimulation of the end-organs in the semicircular canals.

The first portion of the examination consists in the recognition of symptoms which are already present. In the second portion of the examination we produce reactions to stimuli, by which we determine the efficiency of the static labyrinth.

Disturbance in the function of the static labyrinth manifests itself by certain objective signs and subjective symptoms. The objective signs are a spontaneous nystagmus and reaction movements. The subjective symptoms are a sensation of rotation of surrounding objects, and of the body itself, and sometimes nausea and vomiting.

The normal physiological stimuli which are aroused in the static labyrinth during ordinary movements of the body do not elicit any visible reflexes, as far as we can determine by examination. But if these physiological stimuli are increased in intensity there

result reflex movements in the eye muscles, and in the muscles of the body and extremities. The reflexes of the eye muscles are called nystagmus, and those of the muscles of the body and extremities are called reaction movements. Failure to elicit these reflex movements by means of adequate stimuli, means that the end-organs in the semicircular canals have been impaired or destroyed. An increase or a diminution in the ease with which these reflexes can be aroused, as compared with a normal individual, indicates a condition of increased or diminished irritability, respectively, of the end-organs in the semicircular canals. An increase or diminution in the intensity or duration of the reflex aroused may be interpreted in the same way.

We will first consider the second method of examination of the static labyrinth, namely, by arousing reflex movements in the muscles of the eyes (nystagmus), and of the body and extremities (reaction-movements).

A study of the nystagmus reflex is of more value than that of the reaction-movements. When properly elicited, nystagmus is a pure reflex, uninfluenced by the will, whereas in the reaction movements there is usually a considerable voluntary element. Besides, nystagmus can be elicited by a much weaker stimulus than is required to arouse reaction-movements, and therefore entails less discomfort to the patient.

These reflexes for the purpose of testing the function of the semicircular canals may be elicited by four different methods:

1. By rotation (rotation-test).
2. By irrigation of the ear with cold or hot water (caloric test).

3. By means of the galvanic current (galvanic test).

4. By compression or aspiration of the air in the external auditory canal (fistula test).

The actual manner of stimulation of the end-organs in the semicircular canals is probably the same in all four methods. It consists in the production of a movement in the endolymph in the semicircular canals, which causes a displacement of the cupula. The latter drags on the hair-cells. The endolymph movements caused by these tests are much more violent than those which take place in ordinary movements of the body.

In the rotation test, the endolymph movement is caused by the inertia of the endolymph at the beginning of rotation of the body, and the momentum which the fluid possesses, when the rotation of the body ceases. In the caloric test, the endolymph movement is caused by differences in the specific gravity of cold and warm portions of the endolymph. In the galvanic test, the endolymph movement is supposed to be the result of kataphoresis. In the fistula test, the endolymph movement is the result of displacement by direct air-pressure and suction.

Our tests are applied to the semicircular canals only, since we assume that when they are diseased, the vestibule is diseased also. We have no means of experimentally exciting the vestibular end-organs.

The four tests above mentioned can be performed so as to give us either qualitative or quantitative knowledge. Thus we are enabled to determine, first, the presence or absence of any function, and second, if function be present, its degree. The information given by the qualitative tests is very definite. Quantitative tests tell

us whether the labyrinth is in a condition of irritation or of depression, and give us a measure of this impairment of function. Unfortunately, these data are not very reliable, because there are so many inconstant factors which enter into the production of these reflexes. Some of these factors we will mention in the description of the various tests. Many of the factors we are completely ignorant of. However, as our knowledge of the functions of the labyrinth increases, we will learn to control these factors in such a way that our quantitative tests will be, in reality, quantitative.

The value of quantitative tests lies in the fact that they enable us to make an early diagnosis of intra- and para-labyrinthine lesions; to determine the improvement or aggravation of the disease; and finally, for comparison, in scientific publication.

Quantitative determination of the irritability of the static labyrinth is made in one of three ways:

1. By determining the strength of irritation which is necessary to arouse nystagmus. This is called by the Germans the determination of the "Reizschwelle." In the rotation test, the strength of irritation is measured by the number of rotations of the body necessary to arouse an after-nystagmus. In the caloric test, it is measured by the time from the beginning of irrigation to the onset of the nystagmus, or by the amount of water, at a constant temperature, used to bring on nystagmus. In the galvanic test, it is measured by the strength of current necessary to produce nystagmus.

2. By determining the duration of nystagmus, with a constant strength and duration of the irritation. It is questionable whether the duration of the nystagmus is

proportional to the intensity of the irritation. *Barany* thinks that the duration of the nystagmus depends upon the amount of energy which is stored up in the central nuclei of the vestibular nerve. He thinks the stimulus aroused by the irritation causes the centre to unload its store of energy, and that the nystagmus continues until the centre is emptied of its energy.

3. By determining the intensity of the nystagmus, with a constant strength and duration of the irritation. The intensity of the nystagmus aroused by one labyrinth is measured by attempting to neutralize it by means of a nystagmus in the opposite direction, which is elicited from the opposite labyrinth. If one labyrinth is less irritable than the other, there will be a nystagmus toward one or the other side (depending upon the form of stimulation), when the same stimulus is applied simultaneously to both sides. This comparative test can be performed only by the caloric and galvanic methods of irritation, for in the rotation test the irritation of one labyrinth does not oppose, but reenforces that of the opposite labyrinth.

The comparative tests are made either between the two ears of the same person, simultaneously or successively, or between the suspected ear and the ear of a normal individual.

THE ROTATION TEST

If a person is rotated about a vertical axis which passes through his own body there will be elicited certain reflex movements of the eyes and body. These reflexes are the result of endolymph movements in the semicircular canals. The character of the reflexes de-

pends upon the direction of rotation, and the position of the head. The strongest endolymph movement is set up in those semicircular canals which lie in the horizontal plane. The greater the angle which the canals form with the horizontal plane the less will their endolymph contents be acted upon by rotation about a vertical axis. When a canal lies in the vertical plane its contents are not affected at all.

The various canals are tested by placing the head in such a position that the canals to be examined lie in the horizontal plane.

At the beginning of rotation there is an endolymph movement in a direction opposite to the rotation, as a result of the inertia of the fluid. This causes a nystagmus, the slow component of which is in the direction opposite to the rotation of the body. After a few rotations at uniform speed, the endolymph moves at the same rate of speed as the body. It is then at rest in relation to the body, and the nystagmus ceases. When the rotation is stopped, the endolymph, having acquired a certain momentum, keeps on moving for a short time, in the direction in which the body was rotated. This causes an after-nystagmus, the slow component of which is in the direction of rotation; in other words, an after-nystagmus opposite to the direction of the rotation.

To take a concrete example, we will place the head in an upright position. This places the external semicircular canals approximately in the horizontal plane. If the body is rotated to the left there will result, at first, an endolymph movement to the right in both external semicircular canals (Fig. 24). This will cause a hori-

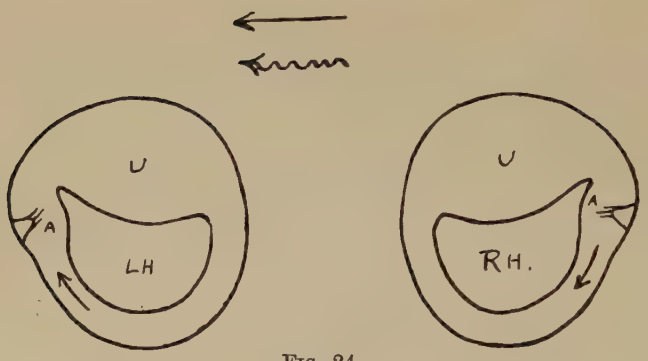

FIG. 24

ROTATION OF BODY TO LEFT, WITH HEAD UPRIGHT

RH—Right external semicircular canal
LH—Left external semicircular canal
U—Utricle
A—Ampulla

The arrow within the canal shows the direction of the endolymph current

The straight arrow, outside of the diagram, indicates the direction of rotation of the body

The wavy arrow indicates the direction of the nystagmus

zontal nystagmus to the left. When the rotation is stopped the endolymph keeps on moving to the left for a short time (Fig. 25). This will give rise to a horizontal after-nystagmus to the right.

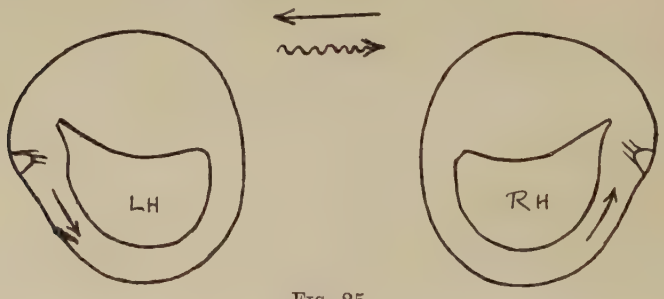

FIG. 25

AFTER-NYSTAGMUS, WHICH OCCURS ON STOPPING, AFTER ROTATION TO THE LEFT

The nystagmus during rotation is in the direction of

the rotation; the after-nystagmus is in a direction opposite to that of the rotation.

In performing the rotation test at least two canals are always acted upon at the same time. As the direction of the endolymph current in the two external semicircular canals is the same, during the rotation of the body, the nystagmus produced by one horizontal canal re-enforces that produced by the other.

In performing the rotation test observation of the nystagmus during turning is attended with such difficulty that we discard it, and rely entirely upon observing the after-nystagmus.

In the succeeding descriptions, therefore, we will disregard the nystagmus during rotation, and consider only the after-nystagmus.

The external semicircular canal does not lie exactly in the horizontal plane, but is tilted about 30° downward and backward, when the head is in the upright position. In order to bring the canal into the horizontal plane for the rotation test, the head should be tilted about 30° forward (Fig. 26). This brings the superior and posterior canals into the vertical plane. The endolymph in these two canals is therefore not influenced by the rotation. As the nystagmus is parallel to the plane of the semicircular canal which causes it, we will have a purely horizontal nystagmus, when the head is tilted 30° forward.

The rotation test can be performed in one of two ways:

1. The person is rotated a certain number of times at a constant rate of speed, and the duration of the after-nystagmus is noted.

2. The number of rotations which is required to cause the onset of an after-nystagmus is noted. In this method the patient is rotated once, and after-nystagmus is watched for. If it does not appear he is rotated twice, and again observed. In this way the number of rotations is increased until an after-nystagmus appears.

In the first method the person is rotated ten times. *Barany* found that ten rotations give the maximum amount of after-nystagmus. If the rotations are increased beyond this number the after-nystagmus diminishes in duration. He also found that in a normal individual ten rotations, at the rate of about 2″ for each rotation, give rise to an after-nystagmus which lasts from 20″ to 40″. There are considerable variations between different individuals, between the two sides of the same individual, and in the same individual on different days.

The rotation test is used in order to compare the two labyrinths. We are enabled to do this because of the fact that in the external canals, stimulation caused by displacement of the cupula toward the utricle is greater than that caused by displacement of the cupula away from the utricle, and in the vertical canals, stimulation caused by displacement of the cupula toward the utricle is less than that caused by displacement of the cupula away from the utricle.

These facts are made use of in the following way:

With the head vertical, or tilted 30° forward, the patient is rotated 10 times to the left, at a constant rate of speed. When the rotation is stopped the endolymph keeps moving to the left for a short while. This causes

an after-nystagmus to the right, lasting 20″ to 40″, if the right labyrinth is normal (Fig. 25). He is then rotated 10 times to the right. On stopping, the endo-

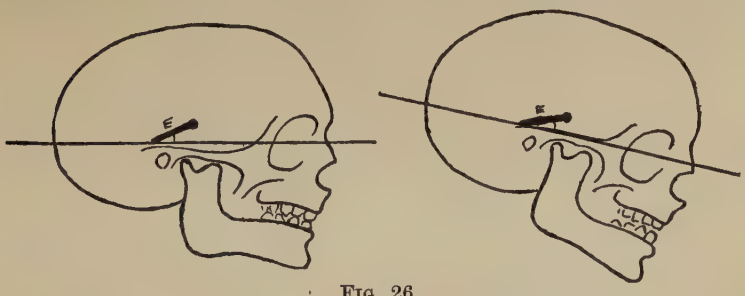

FIG. 26

FIG. 26 (A) FIG. 26 (B)

PLANE OF EXTERNAL SEMICIRCULAR CANAL, WITH HEAD VERTICAL, AND WITH HEAD TILTED 30° FORWARD

E—External semicircular canal

lymph continues to move to the right for a short time. This gives rise to an after-nystagmus to the left, lasting 20″ to 40″, if the left labyrinth is normal (Fig. 27).

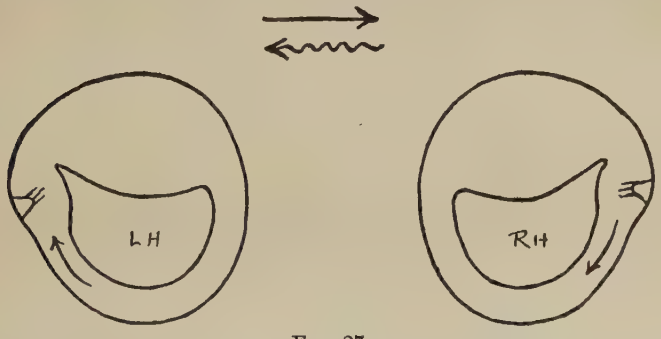

FIG. 27.

AFTER-NYSTAGMUS TO THE LEFT, FOLLOWING ROTATION TO THE RIGHT

Let us now assume that the right labyrinth is not functionating. The results will then be as follows:

After rotating the patient 10 times to the left, the endolymph continues to move to the left for a short

time. This will cause an after-nystagmus to the right. The duration of this after-nystagmus is short, because the displacement of the cupula in the left external canal is away from the utricle (Fig. 28). Occasionally there is no after-nystagmus whatever.

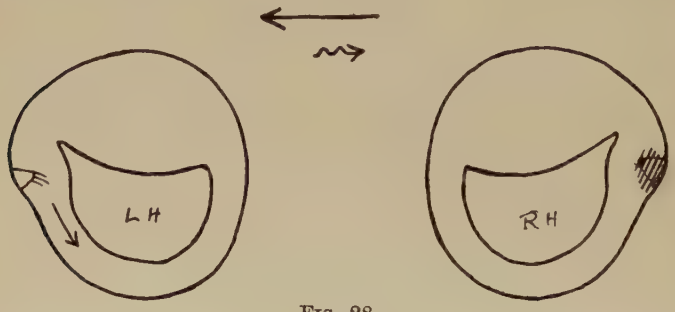

FIG. 28

RIGHT EXTERNAL SEMICIRCULAR CANAL IS NON-FUNCTIONATING. ROTATION TO LEFT CAUSES SHORT AFTER-NYSTAGMUS TO RIGHT

After rotating 10 times to the right the endolymph continues to move to the right for a time. This causes an after-nystagmus to the left, which lasts a comparatively long time, because the cupula in the left semicircular canal has been displaced toward the utricle (Fig. 29).

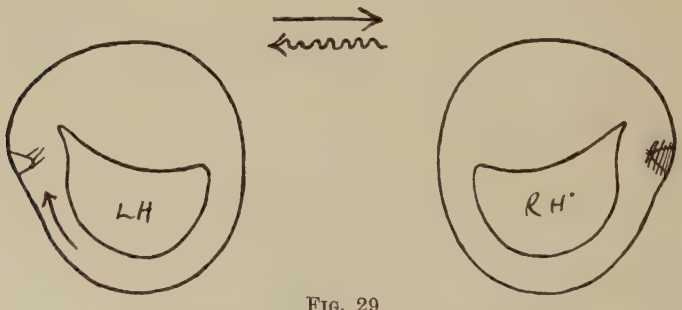

FIG. 29

RIGHT EXTERNAL SEMICIRCULAR CANAL IS NON-FUNCTIONATING. ROTATION TO RIGHT CAUSES LONG AFTER-NYSTAGMUS TO LEFT

The labyrinth on the side opposite to the direction of rotation is stimulated strongest when the rotation is stopped.

The labyrinth on the side toward which the nystagmus is directed is stimulated strongest.

A marked difference in the duration of the after-nystagmus following rotation to the right and to the left indicates that the canal of one side is not functionating. The difference must be more than double in order to be of diagnostic value. Often the difference is even greater than this. The after-nystagmus in one direction may be only 4″ or 5″, while that in the opposite direction is 20″ or 25″.

After-nystagmus in the direction of the functionating canal lasts longer than that in the direction of the non-functionating canal. Rotation toward the destroyed labyrinth causes a longer after-nystagmus than rotation toward the healthy labyrinth. In the above example, with the right labyrinth destroyed, rotation to the right caused a longer after-nystagmus (after-nystagmus to the left) than rotation to the left (after-nystagmus to the right).

The rotation test is performed as follows:

The patient is seated in a revolving chair, with a back and foot-rest. It is advisable to strap him in the chair, as he may fall out during the rotation. In most cases we test only the external semicircular canals by rotation. The head is consequently tilted 30° forward. It is advisable to place ground-glass spectacles before the patient's eyes, according to *Barany,* in order to eliminate fixation, and accommodation with convergence, as these influence the nystagmus. Fixation of

objects in the room has the effect of shortening the duration of the nystagmus. With the eyes in the lateral position, convergence affects the nystagmus in the two eyes in different ways. It increases the nystagmus in one eye and decreases it in the other. In order to eliminate this disturbing influence *Barany* uses ground-glass spectacles. *Bartels* achieves the same result by using strong convex lenses, through which the patient cannot see clearly. They have the added advantage that the observer can see the patient's eyes magnified, through the glasses, and so can better observe the nystagmus.

The patient is rotated 10 times to the right, at a constant rate of speed, the 10 rotations taking about 20″. On stopping, the patient is told to look to the left, and the duration of the after-nystagmus, which in this case is a horizontal nystagmus to the left, is noted by means of a stop-watch. The reason for directing the patient to look to the left is in order to elicit the maximum amount of nystagmus. The nystagmus is always greatest when the eye looks in the direction of the rapid component.

After several minutes of rest the patient is rotated to the left 10 times, at the same rate of speed. On stopping he is directed to look to the right, and the duration of the after-nystagmus, which in this case is a horizontal nystagmus to the right, is noted with the stop-watch.

If the duration of the after-nystagmus is normal in time, and approximately the same in both directions, it is probable that both labyrinths are normal. If the duration of the after-nystagmus in one direction is less

than one-half that of the after-nystagmus in the opposite direction, it is probable that the end-organ in the external semicircular canal of one side is not functionating. The non-functionating labyrinth is on the side of the lesser after-nystagmus, as in rotation, each labyrinth arouses the greater after-nystagmus in its own direction.

In order to test the vertical semicircular canals by means of rotation, they must be brought into the horizontal plane. The superior canal of one side being parallel to the posterior canal of the opposite side, it is possible to bring these two canals into the horizontal

FIG. 30.

plane together. The two superior canals, or the two posterior canals cannot be brought into the horizontal plane together.

The changes in the position of the head, which must be made in order to make the vertical canals horizontal, can be understood very readily by manipulating two oblong pieces of cardboard, cut half way down the middle, and joined together at right angles to each other, as in Figs. 30 and 31.

Place the cards in the vertical plane, so that each one is at 45° to the sagittal and transverse planes of the head. The anterior halves of the two cards lie in the planes of the two superior semicircular canals, and the

posterior halves in the planes of the two posterior semicircular canals. By rotating these cards in various directions, it can readily be determined how it will be necessary to rotate the head in order to bring the individual vertical canals into the horizontal plane.

If the head is bent 90° forward all four vertical semicircular canals lie in planes 45° to the horizontal (Fig. 32). If the head is now rotated so that the face looks

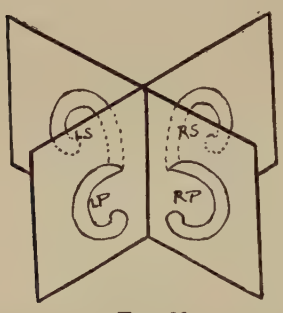

FIG. 31

REPRESENTATION OF PLANES OF VERTICAL SEMICIRCULAR CANALS

RP—Right posterior semicircular canal
RS—Right superior semicircular canal
LP—Left posterior semicircular canal
LS—Left superior semicircular canal

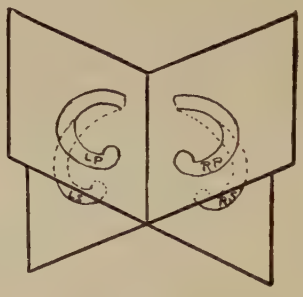

FIG. 32

PLANES OF VERTICAL CANALS, WITH HEAD TILTED 90° FORWARD

RP—Right posterior semicircular canal
RS—Right superior semicircular canal
LP—Left posterior semicircular canal
LS—Left superior semicircular canal

toward the right shoulder, the right superior and the left posterior canals lie in the horizontal plane (Fig. 33).

If, with the head in this position, the patient is rotated to the left 10 times, the endolymph, on account of its momentum, will continue to move to the left, in the right superior and the left posterior canals, after

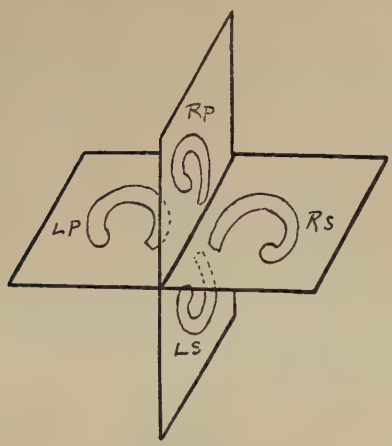

FIG. 33

PLANES OF VERTICAL CANALS WITH HEAD TILTED 90° FORWARD AND FACE TURNED TO RIGHT SHOULDER

the rotation is stopped. This will give rise to a rotatory after-nystagmus to the right (Fig. 34). A rotatory nystagmus to the right means a nystagmus in which the upper end of the vertical meridian of the iris moves downward and to the right during the rapid phase.

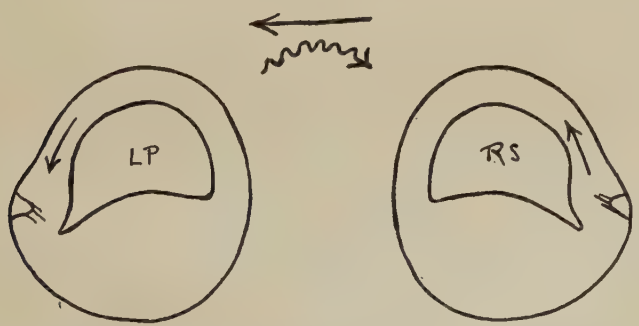

FIG. 34

RIGHT SUPERIOR AND LEFT POSTERIOR CANALS IN HORIZONTAL PLANE, WITH HEAD 90° FORWARD, AND FACE TURNED TO RIGHT SHOULDER. ROTATORY AFTER-NYSTAGMUS TO RIGHT, FOLLOWING ROTATION OF BODY TO LEFT

In the right superior canal the cupula is displaced away from the utricle, and in the left posterior canal it is displaced toward the utricle. Since, in the vertical canals, the crista is stimulated more powerfully when the cupula is displaced away from the utricle than when it is displaced toward the utricle, the rotatory after-nystagmus to the right is elicited principally by the right superior canal. In this regard, therefore, the conditions in the vertical canals are similar to those in the horizontal canals.

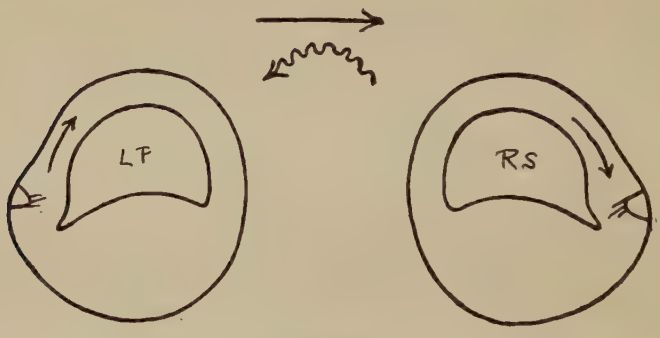

FIG. 35

POSITION OF HEAD SAME AS IN PRECEDING DIAGRAM. ROTATORY AFTER-NYSTAGMUS TO LEFT, FOLLOWING ROTATION OF BODY TO RIGHT

If, with the head in the above-described position, the patient is rotated to the right 10 times, there will result a rotatory after-nystagmus to the left (Fig. 35). This nystagmus is elicited principally by the left posterior canal, as in this canal the cupula is displaced away from the utricle, while in the right superior canal the cupula is displaced toward the utricle.

If the right superior canal has a non-functionating end-organ, rotation to the right will cause a longer rota-

tory after-nystagmus (to the left) than rotation to the left (after-nystagmus to the right) (Figs. 36 and 37).

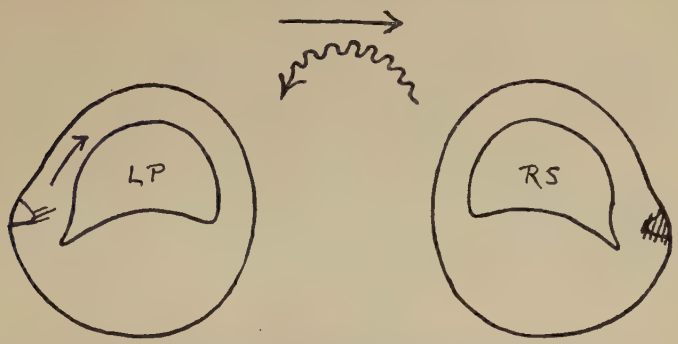

FIG. 36

RIGHT SUPERIOR CANAL IS NOT FUNCTIONATING. LONG ROTATORY AFTER-NYSTAGMUS TO THE LEFT FOLLOWS ROTATION OF THE BODY TO THE RIGHT

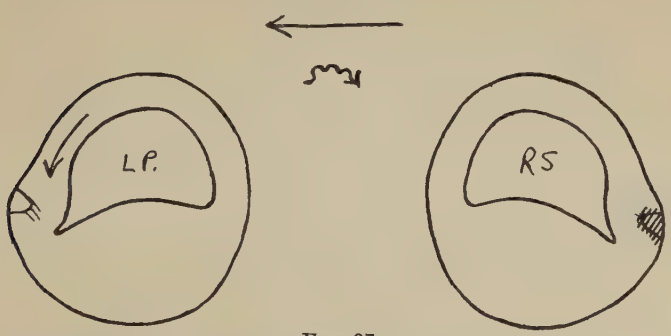

FIG. 37

RIGHT SUPERIOR CANAL IS NOT FUNCTIONATING. SHORT ROTATORY AFTER-NYSTAGMUS TO THE RIGHT FOLLOWS ROTATION OF THE BODY TO THE LEFT

In this regard the vertical canals react like the horizontal canals.

If, with the head bent 90° forward, the face is turned to the left shoulder, the left superior and the right posterior canals are brought into the horizontal plane

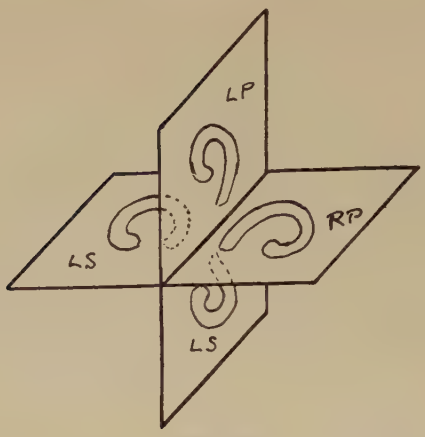

Fig. 38

Planes of Vertical Canals With Head Tilted 90° Forward, and Face Turned to Left Shoulder

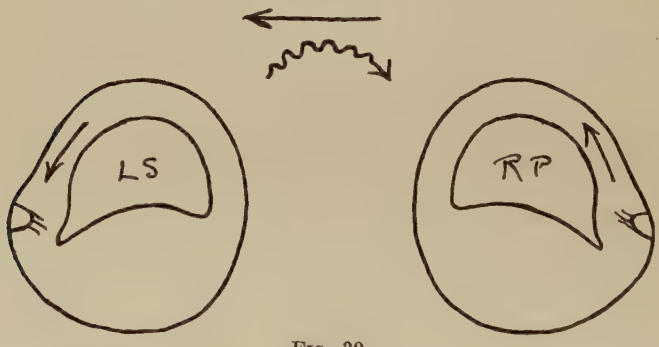

Fig. 39

Right Posterior and Left Superior Canals in Horizontal Plane With Head 90° Forward, and Face Turned to Left Shoulder. Rotatory After-Nystagmus to Right, Following Rotation of Body to Left

(Fig. 38). If, with the head in this position, the patient is rotated 10 times to the left, the endolymph in the left superior and right posterior canals will continue to flow for a short time to the left, after the rotation has stopped. This will give rise to a rotatory af-

ter-nystagmus to the right, the nystagmus being elicited principally by the right posterior canal, since the cupula in this canal is displaced away from the utricle (Fig. 39).

If the patient is rotated to the right, there will be a rotatory after-nystagmus to the left. The nystagmus is elicited chiefly by the left superior canal (Fig. 40).

If the right posterior canal has a non-functionating end-organ, rotation to the right will cause an after-nystagmus (to the left) of longer duration than rota-

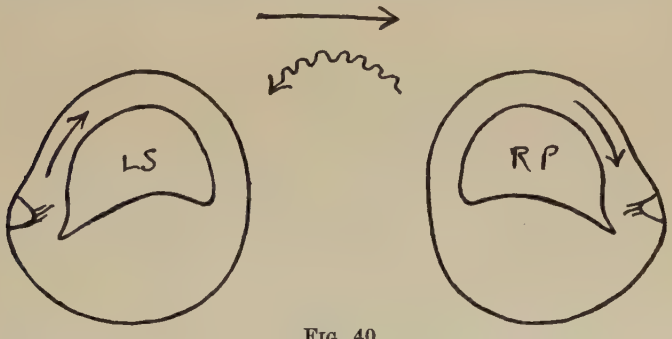

FIG. 40

ROTATORY AFTER-NYSTAGMUS TO LEFT, FOLLOWING ROTATION TO RIGHT

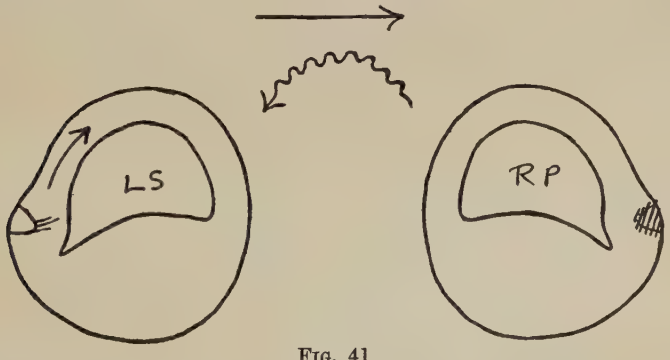

FIG. 41

RIGHT POSTERIOR CANAL IS NOT FUNCTIONATING. LONG ROTATORY AFTER-NYSTAGMUS TO LEFT, FOLLOWING ROTATION OF BODY TO RIGHT

tion to the left (rotatory after-nystagmus to the right) (Figs. 41 and 42). All three semicircular canals, therefore, arouse a stronger after-nystagmus toward their own side, than toward the opposite side.

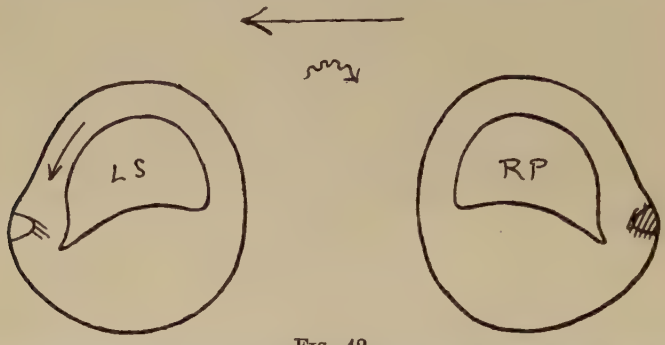

FIG. 42

RIGHT POSTERIOR CANAL IS NOT FUNCTIONATING. SHORT ROTATORY AFTER-NYSTAGMUS TO RIGHT, FOLLOWING ROTATION OF BODY TO LEFT

Tho rotatory after-nystagmus produced by rotation does not last as long as the horizontal after-nystagmus produced in this way.

If, instead of bending the head forward, it is bent backward 90°, and the face turned to the right, the right superior and left posterior canals come to lie in the horizontal plane (Figs. 43 and 44).

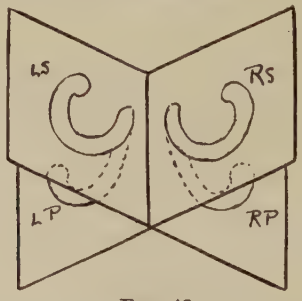

FIG. 43

PLANES OF VERTICAL CANALS, WITH HEAD TILTED 90° BACKWARD

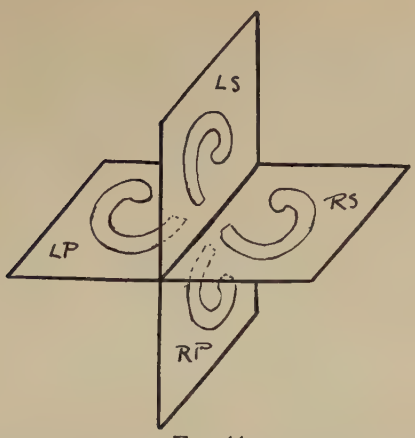

FIG. 44

PLANES OF VERTICAL CANALS, WITH HEAD TILTED 90° BACKWARD, AND FACE TURNED TOWARD THE RIGHT SHOULDER

After rotation to the left, the endolymph in the right superior and left posterior canals continues to flow to the left for a short time. But the rotatory after-nystagmus is to the left, because the vertical canals are placed in a position opposite to that in which they lay when the head was bent forward (Fig. 45). This nys-

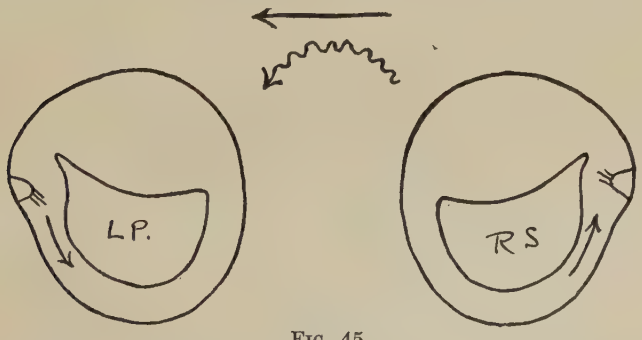

FIG. 45

HEAD BENT 90° BACKWARD, AND FACE TURNED TOWARD THE RIGHT SHOULDER. ROTATORY AFTER-NYSTAGMUS TO LEFT, FOLLOWING ROTATION TO THE LEFT. NYSTAGMUS ELICITED PRINCIPALLY BY LEFT POSTERIOR CANAL

tagmus is elicited principally by the left posterior canal. Here, again, the after-nystagmus is aroused mainly by the canal on its own side. If the head is rotated to the right, there will follow a rotatory after-nystagmus to the right, aroused mainly by the right superior canal (Fig. 46).

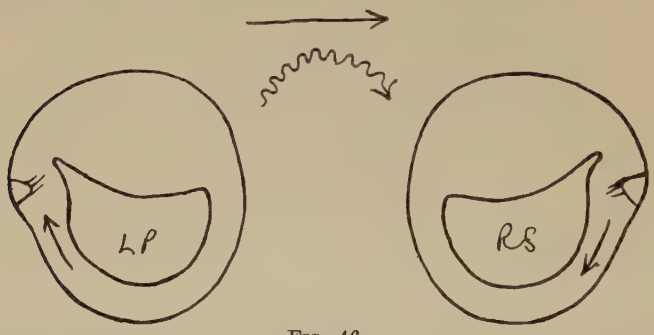

FIG. 46

ROTATORY AFTER-NYSTAGMUS TO RIGHT, FOLLOWING ROTATION TO RIGHT. NYSTAGMUS ELICITED PRINCIPALLY BY RIGHT SUPERIOR CANAL

If, with the head turned 90° backward, the face is turned to the left, the left superior and right posterior

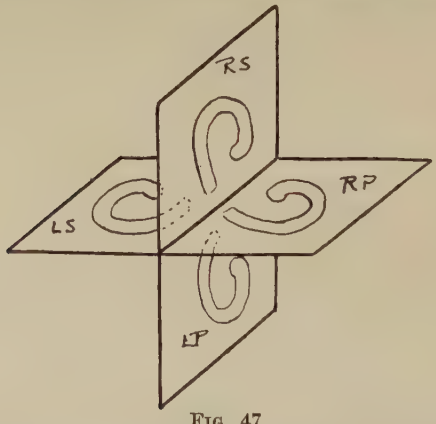

FIG. 47

PLANES OF VERTICAL CANALS, WITH HEAD TILTED 90° BACKWARD, AND FACE TURNED TOWARD THE LEFT SHOULDER

canals come to lie in the horizontal plane (Fig. 47). If the patient is now rotated to the left there follows a rotatory after-nystagmus to the left, elicited mainly by the left superior canal (Fig. 48) .

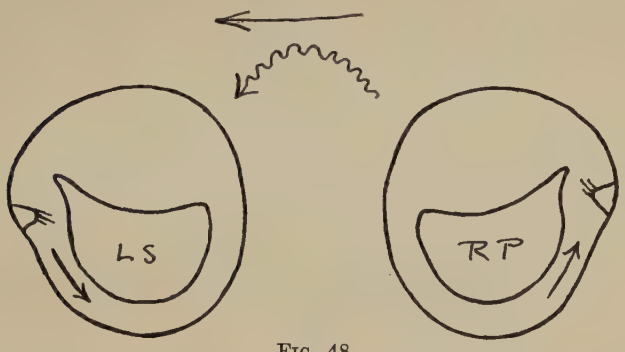

Fig. 48

Head Bent 90° Backward, and Face Turned Toward Left Shoulder. Rotatory After-Nystagmus to Left, Following Rotation to Left. Nystagmus Elicited Chiefly by Left Superior Canal

If the patient is rotated to the right there results a rotatory after-nystagmus to the right, which is aroused principally by the right posterior canal (Fig. 49).

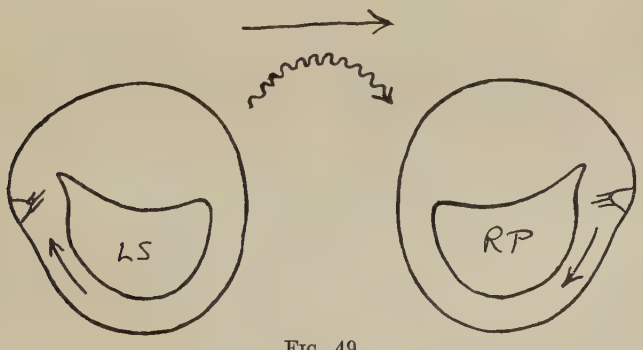

Fig. 49

Rotatory After-Nystagmus to the Right, Following Rotation to the Right. Nystagmus Elicited Chiefly by Right Posterior Canal

When the head is bent forward 90° all four vertical canals lie at 45° to the horizontal plane. If the patient is now rotated all four vertical canals are acted upon to the same degree, and there results a rotatory nystagmus, which is due to the combined effects of all four vertical canals. After rotation to the right, there is a rotatory after-nystagmus to the left, and after rotation to the left, a rotatory after-nystagmus to the right. When the head is bent backward 90°, the same results are obtained, except that the nystagmus is in the opposite direction; i. e., after rotation to the right, there follows a rotatory after-nystagmus to the right, and after rotation to the left, there follows a rotatory after-nystagmus to the left.

If the head is tilted 90° to one shoulder, and the patient rotated, there results a vertical nystagmus. In this position all four vertical canals are also 45° to the horizontal plane. The reason we get a vertical nystagmus in this position instead of a rotatory nystagmus, is probably because the relative positions of the individual canals to the horizontal and vertical planes, when the head is tilted toward the shoulder, is not the same as when the head is tilted forward.

When the head is bent 90° to the right shoulder, and the patient is rotated to the right, there follows a vertical after-nystagmus downward. If he is turned to the left, there follows a vertical after-nystagmus, upward.

With the head bent 90° to the left shoulder, and the patient rotated to the right, there follows a vertical after-nystagmus, upward. When the patient is rotated to the left, there is a vertical after-nystagmus, downward.

If the head is held between the vertical position and 90° forward, all six semicircular canals are affected by rotation, and there results a combination of rotatory with horizontal nystagmus. The more nearly vertical the head is held the more will the horizontal nystagmus predominate over the rotatory, and the more the head is bent forward, the more will the rotatory nystagmus predominate over the horizontal.

If the head is held between the vertical and 90° backward, there will be a combined rotatory and horizontal nystagmus, but the rotatory component will be in a direction opposite to that of the horizontal component. For instance, after rotation to the right, there will follow a horizontal after-nystagmus to the left, combined with a rotatory after-nystagmus to the right. If the patient looks toward the left the rotatory element will disappear, and there will be a pure horizontal nystagmus to the left. If he looks toward the right the horizontal element will disappear, and there will be a pure rotatory nystagmus to the right.

From the above facts the following general law was deduced by *Barany:*

"In rotating a person about a vertical axis, the character of the nystagmus is indicated by the line in which the horizontal plane cuts the cornea."

When spontaneous nystagmus is present the interpretation of the results of the rotation test is much more difficult. If the spontaneous nystagmus is present only when the patient looks in one direction, we can use *Barany's* "blickfixator," or *Brünings'* modification of this instrument, which he calls the "otogoniometer." The "blickfixator" consists of a head-band, to the front

of which is attached a projecting horizontal rod, about 15 cm. in length, which can be moved from side to side. At the end of the horizontal rod is a short vertical rod, to the lower end of which is attached a small knob. The patient is told to fix his eyes upon the knob, and the horizontal rod is turned until it is in such a position, that when the patient fixes the knob the spontaneous nystagmus disappears.

After the patient is rotated he is told to look at the knob again. In this way the spontaneous nystagmus does not obscure the nystagmus which is elicited by the rotation test.

When the spontaneous nystagmus is present in every position of the eyes, we must try to estimate the effect upon the spontaneous nystagmus by the nystagmus produced by rotation. If, for instance, there is a spontaneous nystagmus to the right, and we rotate the patient to the left, there will result, upon cessation of the rotation, an after-nystagmus to the right. For a time the spontaneous nystagmus will be intensified. When the after-nystagmus has ceased the spontaneous nystagmus will resume its normal intensity. We measure the length of time during which the nystagmus was intensified. On rotating the patient to the right there will follow an after-nystagmus to the left. This will diminish the intensity of the spontaneous nystagmus for a time. We now compare the length of time during which the spontaneous nystagmus was intensified, with the length of time during which it was weakened, and draw our conclusions in regard to the functional activity of the two labyrinths therefrom.

THE CALORIC TEST

Schmiedekam and *Hensen* discovered, in 1868, that cold water poured into the ear caused vertigo and vomiting. *Cohn* and *Urbantschitsch* discovered that cold and hot water, poured into the ear, caused nystagmus. But *Barany* made a systematic study of the principles of the caloric reaction, and made of it a valuable means for the estimation of the functional activity of the semicircular canals.

He found that if the head is held in an upright position, and cold water is poured into one ear, there will result a combined rotatory and horizontal nystagmus toward the opposite ear. If water which is warmer than the temperature of the body is poured into the ear there will result a combined rotatory and horizontal nystagmus toward the irrigated ear. If water of body-temperature is used no nystagmus results. If the head is rotated forward 180°, so that the vertex points downward, the conditions are reversed. Cold water now causes nystagmus toward the irrigated ear, and warm water, away from the irrigated ear. Thus it is seen that the direction and character of the caloric nystagmus depend on the temperature of the water and the position of the head. If cold or hot water elicit no nystagmus the labyrinth on that side is non-functionating, or has impairment of its function.

Earlier observers thought that the nystagmus elicited by cold and hot water was due to the pressure of the water. But *Barany* proved that this could not be the case, for the following reasons:

1. The reaction can be elicited in favorable cases (for instance, in an epidermatized radical cavity), by means

of water dropped from a medicine dropper. In such a case there is practically no pressure whatever.

2. When the water is of body temperature there is no reaction.

3. The nystagmus is in one direction when cold water is used, and in the opposite direction when warm water is used.

4. Changing the position of the head changes the direction of the nystagmus.

Barany ascribes the reaction to an endolymph movement produced by the difference in specific gravity of cold and warm portions of the endolymph. When cold water is poured into the ear the cold penetrates through the inner tympanic wall to the labyrinth. The anterior portion of the external semicircular canal, and the ampullated end of the superior canal, lie nearest to the middle-ear cavity, and consequently the endolymph in these portions of the semicircular canals is chilled first (Fig. 50).

Fig. 50

Relations of Semicircular Canals to Tympanic Cavity

TC—Tympanic cavity
S—Ampulla of superior canal
E—Ampulla of external canal
P—Ampulla of posterior canal

The arrow indicates the direction of the current of cold or hot water

If cold water is poured into the right ear the endolymph in the ampulla of the right superior semicircular

canal becomes chilled. As the diminution in temperature makes it heavier it drops toward the utricle (Fig. 51). The endolymph in the outer limb of the supe-

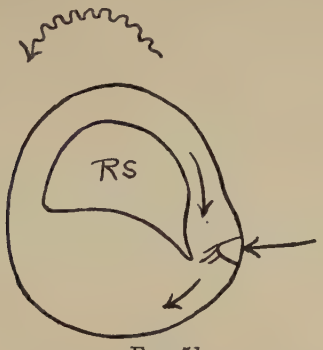

Fig. 51

Right Superior Canal. Straight Arrow on Outside Indicates Point of Application of Cold Water. Arrows Within the Canal and Utricle Indicate Direction of Endolymph Current. Wavy Arrow Indicates Direction of Nystagmus

rior canal follows the chilled endolymph, which has dropped, and it, in turn, becomes chilled by the cold water in the middle ear. In this way an endolymph current is set up in the superior canal, toward the utri-

Fig. 52

Diagram Illustrating the Influence Which Endolymph Current in Superior Canal Exerts on Endolymph in Posterior Canal, as Result of Common Limb

S—Superior canal *P*—Posterior canal

U—Utricle

cle. The current continues until all of the fluid is of the same temperature.

The endolymph movement in the superior canal sets into motion the fluid in the posterior canal, because both canals have a common limb (Fig. 52). The endolymph movement in both the vertical canals is toward the utricle. This gives rise to a rotatory nystagmus toward the opposite side, i. e., toward the left.

The horizontal canal is tilted backward about 30°. The endolymph in its anterior portion becomes chilled, and flows downward and backward. This gives rise to an endolymph movement away from the utricle (Fig. 53), which results in a horizontal nystagmus, also to the

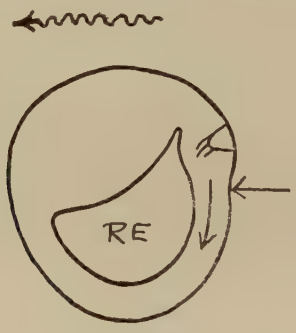

Fig. 53

Right External Canal. Straight Arrow on Outside Indicates Point of Application of Cold Water

opposite side, i. e., to the left. As the horizontal canal is tilted only 30° from the horizontal plane, and the vertical canals are almost vertical, when the head is in the upright position, the horizontal element of the nystagmus is much less conspicuous than the rotatory element.

When hot water is used the warmed endolymph is

pushed up by the cooler fluid, and the current is in the opposite direction. This gives rise to a rotatory and horizontal nystagmus toward the same side.

A portion of the external semicircular canal lies exposed in the aditus. Here the endolymph is most accessible to the cold water which is poured into the ear. If it were possible to bring the external semicircular canal to lie in the vertical plane, an endolymph movement could be set up in this canal long before it would be aroused in the other two canals. This can easily be done as follows; the external semicircular canal makes an angle of 30° with the horizontal plane, when the head is upright. If the head is tilted backward 60° the external canal lies in the vertical plane (Fig. 54).

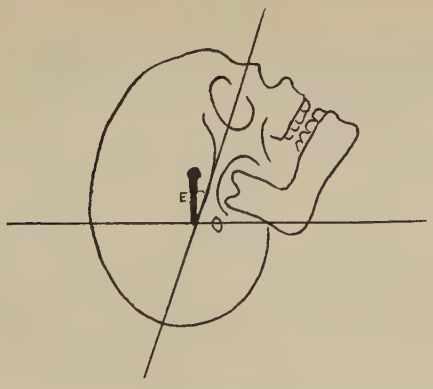

Fig. 54
"Optimum Position" for Caloric Test on External Semicircular Canal
E—External semicircular canal

Brünings found that with the head in this position the external canal responds very quickly to the caloric test. The position may be rendered still more favorable by the following manœuvre; after tilting the head back-

ward 60°, rotate it 45° about its antero-posterior axis, so that the ear to be irrigated is depressed toward the shoulder of that side. If the right ear is being examined, that ear should be depressed toward the right shoulder (Fig. 55). The result of this manœuvre is to

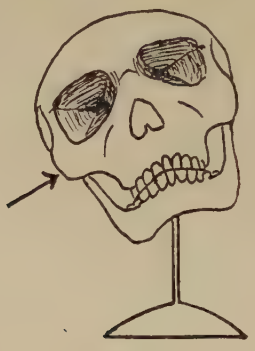

FIG. 55

BRÜNINGS' "OBLIQUE OPTIMUM POSITION" FOR THE RIGHT EXTERNAL SEMICIRCULAR CANAL. HEAD TILTED 60° BACKWARD AND 45° TOWARD RIGHT SHOULDER

rotate the external canal about an axis at right angles to its plane, so that the point of application of the cold water is at a greater distance above the bottom of the column of endolymph; in other words, the fall of the endolymph is greater (Fig. 56).

This position of the head *Brünings* calls the "oblique optimum position" for the external semicircular canal.

With the head in this position, an almost pure horizontal nystagmus to the opposite side is obtained with cold water, and to the same side with hot water.

With the head bent 30° forward, or 120° backward, the external semicircular canal is brought into the horizontal plane. In either of these positions the caloric reaction gives an almost pure rotatory nystagmus.

The advantages in using the optimum position of the external semicircular canal, in performing the caloric test, are the following:

1. The reaction is rapidly obtained with water which is not very cold, so that the amount of discomfort to the patient is reduced to a minimum.

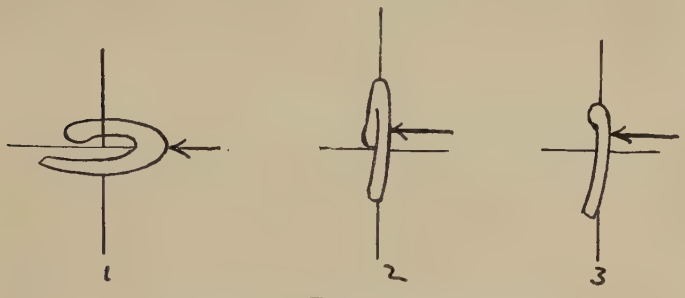

FIG. 56

1—POSITION OF EXTERNAL CANAL IN "PESSIMUM POSITION" FOR CALORIC TEST

2—POSITION OF EXTERNAL CANAL IN "STRAIGHT OPTIMUM POSITION" FOR CALORIC TEST

3—POSITION OF EXTERNAL CANAL IN "OBLIQUE OPTIMUM POSITION" FOR CALORIC TEST

Arrow indicates point of application of cold water

2. As soon as the nystagmus is aroused it can be checked by throwing the head forward 90°, i. e., 30° forward from the vertical position of the head (the so-called pessimum position of the external canal), thus cutting short the vertigo.

3. It helps in an accurate quantitative estimation of the caloric irritability.

4. It allows a separate examination of the external and the vertical canals. This is done as follows:

The head is placed in the oblique optimum position, i. e., 60° backward and 45° toward the shoulder of the side to be examined. Cold water is allowed to flow into

the ear. If no nystagmus results the external canal is non-functionating. The head is now thrown forward into the pessimum position, i. e., 30° forward from the upright position. If there results a rotatory nystagmus in this position the vertical canals are intact. In this way a circumscribed labyrinthitis confined to the external semicircular canal, resulting in the loss of its function, can be determined. *Brünings* was able to make this diagnosis in several cases.

If there is a positive reaction with the head in the optimum position, there is no need to test for the reaction in the vertical canals; for an isolated labyrinthitis in the vertical canals is practically unknown.

With failure to obtain a reaction in the optimum position, after a reasonable time, it is usually unnecessary to continue the irrigation, in order to obtain the rotatory nystagmus in the pessimum position, the endolymph being already sufficiently cooled to give the reaction. After discontinuing the irrigation, a horizontal nystagmus, with the head in the optimum position, is immediately checked, and a rotatory nystagmus substituted, when the head is thrown into the pessimum position.

The caloric test has the advantage over the rotation test, in that by it each labyrinth can be tested separately.

Ordinarily the caloric test is performed with cold water. The use of hot water is much more disagreeable to the patient, as it is necessary to use water of at least 110° F. in order to elicit a "hot water nystagmus." Hot water is of use chiefly in those cases where a spontaneous nystagmus is present. If there is spon-

taneous nystagmus to the left, and we suspect disease in the right labyrinth, the right ear is irrigated with hot water. If the right labyrinth is still functionating, the caloric nystagmus, which is aroused by the hot water, and which is directed toward the right, will neutralize or overcome the spontaneous nystagmus toward the left. If the spontaneous nystagmus is unaffected the labyrinth is non-functionating.

If the spontaneous nystagmus is not present in every position of the eyes, *Barany's* "blickfixator" or *Brünings'* "otogoniometer" may be used. By means of either of these instruments the eyes are held in a position in which there is no spontaneous nystagmus. Cold water is then used to elicit a caloric nystagmus.

Brünings has attempted to estimate the caloric irritability of the labyrinth, quantitatively. In this he was more or less successful only in normal cases. In order to make a quantitative caloric test the following factors must be made constant:

1. The temperature of the water.
2. The rate of speed of the flow of the water.
3. The direction of the current of water in the external auditory canal.
4. The positions of the semicircular canals, i. e., the position of the head.
5. The position of the eyes.

Brünings was enabled to make these factors fairly constant by means of his "otocalorimeter" and "otogoniometer." The otocalorimeter (Fig. 57) consists of two glass vessels attached to an upright board, one above the other. They are connected by means of rubber tubing, in the course of which is interpolated a

double-current ear-tip. In the upper vessel are placed a thermometer and a funnel, both of which are inserted into air-tight plugs. Through the funnel water is poured into the vessel, and kept at a constant temperature. The double current tip is inserted into the ear to

Fig. 57

Brünings' Otocalorimeter

a certain depth, beyond which it cannot penetrate. The return flow passes into the lower vessel, which is also air-tight, and graduated. From these graduations the amount of water used is read off. The rate of speed of the flow and the pressure are constant in this instrument.

The positions of the semicircular canals and the positions of the eyes are fixed by means of the otogoniometer (Fig. 58). This instrument consists of a head-

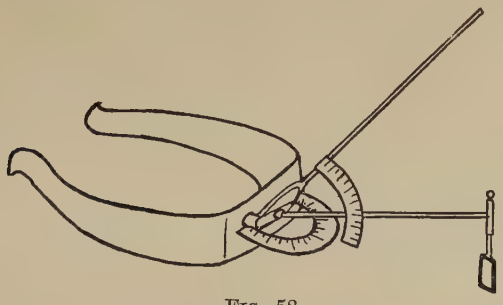

FIG. 58

BRÜNINGS' OTOGONIOMETER

band, to the front of which are attached, by movable joints, two rods. The upper rod moves in a vertical plane, and the lower in a horizontal plane. The upper rod has a graduated arc attached to it, near its junction with the head-band. The horizontal rod has a small mirror attached to its distal end. This rod moves across the upper surface of a graduated arc, which is attached to the front of the head-band. The upper rod is used to determine the position of the external semicircular canal. With the head upright, this rod is raised through an arc of 30°. The rod now corresponds to the plane of the external semicircular canal. The head is then bent backward until the rod is exactly vertical. In this position of the head the external semicircular canal is vertical.

The lower rod, with the mirror at the end, is used to fix the position of the eyes. The rod is rotated 50° to one or the other side of the median line, according to which ear is being tested. The patient is then directed

to look into the small mirror, which is rotated from side to side, until the patient can see some object in the room reflected in the mirror. In this way the accommodation and convergence which occur in the use of *Barany's* "blickfixator" are avoided. If the patient looks more than 50° to the side there often occurs a spontaneous physiological nystagmus, which is very disturbing during the examination.

The quantitative caloric test is performed as follows: the head is placed in the optimum oblique position for the external semicircular canal. The eyes are directed by means of the otogoniometer 50° toward the opposite side. Water of a temperature of 27° C. or 80° F. is allowed to flow into the ear from the otocalorimeter. As soon as nystagmus sets in the flow of water is stopped, and the amount used is read off from the lower receptacle. The rate of flow is such that 300 cc of water are used up in 3 minutes.

Brünings found that in normal ears the average amount of water at 27° C. used to arouse nystagmus was 70 cc. The time required was between ½ and 1 minute. In cases where water of 27° C. does not elicit nystagmus in 3 or 4 minutes, colder water is used. In pathological cases the quantitative caloric test does not give very reliable data, because, in addition to changes in the internal ear, and the retrolabyrinthine paths, the time of onset of the nystagmus is influenced by changes in the temperature-conductivity to the internal ear. These disturbances of temperature-conductivity may be due to:

1. Defects in the drum-membrane. A small perforation does not influence the temperature-conductivity

much. But where most of the drum-membrane is gone, or in an epidermatized radical cavity, nystagmus can be aroused in half or even quarter of the usual time.

2. Stenosis of the external auditory canal.
3. Granulations in the middle ear or canal.
4. Cholesteatoma.

Any of these three conditions, or all three together, may delay the onset of the nystagmus considerably.

5. Inflammatory hyperæmia of the middle-ear. In acute inflammation of the middle-ear, the large quantity of blood which passes through the mucous membrane of the middle-ear delays the chilling of the endolymph by the cold water considerably.

Kiproff and *Beck,* as a result of a series of tests, came to the conclusion that the time required to bring on caloric nystagmus depends upon extra-labyrinthine conditions, whereas the duration of the nystagmus depends upon labyrinthine or central conditions.

Kiproff examined a large number of normal and diseased ears by the caloric method, and found that where the labyrinth was not involved the duration of the nystagmus was the same in all cases (with an equal intensity of stimulus), but the time of onset varied considerably in the different pathological conditions.

His tests were made as follows: water of 30° C. or 86° F. was allowed to run into the ear, with the eyes directed toward the opposite side. At the moment of onset of the nystagmus the eyes were directed straight ahead, and the irrigation continued until nystagmus began in this position of the eyes. Then the irrigation was stopped, and the eyes turned back to the first position. The time from the onset of the nystagmus in the

oblique position to the cessation of the nystagmus was noted.

He found that the onset of the nystagmus was most rapid in the cases of chronic suppuration with total destruction of the membrana tympani, and in healed radicals. The next most rapid were the normal ears. Less rapid was the onset of the nystagmus in the chronic suppurative ears with granulations, cholesteatoma, etc. It was slowest in acute middle ear suppurations. However, the duration of the nystagmus was about 2 minutes in all of the cases.

Beck examined a number of acute suppurative ears by means of the caloric test, each day, from the onset of the disease until they healed. He found that as the inflammation subsided the time of onset of the nystagmus diminished, but the duration of the nystagmus remained the same at all times. By the use of adrenalin in the ear, in these cases, he was also able to diminish the time of onset of the nystagmus.

Ruttin makes a quantitative caloric test by comparing the diseased with the sound ear of the same person. He has constructed an apparatus whereby both ears are irrigated simultaneously, with water of the same temperature, at the same rate of flow. If both labyrinths are normal there will be no nystagmus, for the nystagmus from the right labyrinth neutralizes that from the left. If the right labyrinth is less irritable than the left there will result a nystagmus to the right (with cold water).

In some cases, as, for instance, where there is a dry perforation in the drum-membrane, or in a recently operated radical mastoid, it is inadvisable to use cold

water. In such a case cold air can be substituted for the water. *Aspissoff, Bloch* and *Ruttin* have devised apparatuses for this purpose.

THE GALVANIC TEST

Up to the present the galvanic test has not proven to be of very much value for the diagnosis of labyrinthine disease. The reason for this is that even after the destruction of the labyrinth the galvanic current still elicits nystagmus, by direct stimulation of the vestibular nerve.

In performing the galvanic test both labyrinths may be stimulated together, the anode or positive pole being placed over one ear and the kathode or negative pole being placed over the other; or one labyrinth may be stimulated alone, one pole being placed over one ear, and the other pole over some indifferent part of the body, such as the sternum, arm, hand, etc.

When both ears are stimulated together a very weak current is sufficient to elicit nystagmus, as the stimulation from one labyrinth re-enforces that of the other. But for clinical purposes this method is useless, for it does not differentiate between the functional conditions of the two labyrinths. When one pole is placed over one ear and the other pole over the sternum or some other part of the body, a stronger current is necessary in order to elicit nystagmus.

The galvanic test is performed as follows: Two moistened sponge electrodes are used, one being placed over the sternum or in the hand, and the other directly in front of or behind the ear to be examined. The gal-

vanic current is then turned on, the current being gradually increased in strength until a nystagmus is elicited. The strength of current is read from the milliamperemeter, which is attached to the instrument. The strength of current necessary to arouse nystagmus is a measure of the irritability of the labyrinth. In turning off the current it should be gradually weakened, and not turned off suddenly, in order not to shock the patient unnecessarily.

The character of the nystagmus is a combination of rotatory and horizontal elements, indicating that all three semicircular canals are stimulated together. It has thus far been found impossible to stimulate one semicircular canal alone by the galvanic current, even when the canals have been dissected out and both electrodes placed over one canal.

The direction of the nystagmus depends upon which pole is placed over the ear. When the kathode or negative pole is placed over the ear, the nystagmus is toward that side. When the anode or positive pole is placed over the ear, the nystagmus is toward the opposite side. The nystagmus is in the direction of the flow of the galvanic current. The current flows from the positive to the negative pole. When the kathode is placed over the ear the current leaves the body at that point. The direction of the current corresponds with the direction of the nystagmus, which is toward the examined ear. When the anode is placed over the ear the current enters the body at that point. The direction of the current corresponds with the direction of the nystagmus, which is away from the examined ear.

When the anode is placed over one ear and the

kathode over the other there is nystagmus toward the kathode.

When one pole is placed over each ear a current of 2 to 5 *ma* is sufficient to arouse nystagmus. When only one labyrinth is examined at a time a current of 5 to 10 *ma* is necessary.

The position of the head has no influence on the character or direction of the nystagmus in this test.

When the labyrinth, as well as the vestibular nerve are destroyed, the galvanic current does not cause any nystagmus. However, if the vestibular nerve is not yet degenerated, it will respond to the stimulation.

If the anode is placed over the sternum, and two sponges are attached to the kathode, one of which is placed before each ear, there will be no nystagmus, if both labyrinths are normal. If one labyrinth is destroyed, or if its excitability is diminished, there will result a nystagmus toward the sound ear. If a double anode be placed before the ears the nystagmus will be toward the diseased ear. However, in order to elicit a nystagmus with a double electrode, a very strong current is necessary. In fact, the required current may be so strong as to cause unendurable pain to the patient.

The actual method of stimulation of the end-organs in the labyrinth by means of the galvanic current is not yet positively known. The most plausible hypothesis is that laid down by *Brünings,* which is as follows; the galvanic current sets up a kataphoretic current in the endolymph, or the cupula is set into motion directly by the kataphoresis. As a result of this movement of the endolymph or cupula, the hair-cells are stimulated.

Kataphoresis is an electro-endosmosis. It is a mo-

tion which is produced in liquids by the galvanic current, the fluid moving from one pole to the other. Solid particles which are present in the fluid are also set into motion. On the basis of this theory it would seem that the kataphoretic movement in the endolymph is away from the kathode and toward the anode.

This theory brings galvanic stimulation into harmony with the mechanical methods of stimulating the semicircular canals, namely rotation, cold and heat, and compression and aspiration.

All of the phenomena of galvanic irritation can be explained by this theory; but it is still a theory. It has not been proven.

Many observers claim that there is a direct stimulation of the vestibular nerve by the galvanic current, as the reaction can be obtained after complete destruction of the semicircular canals. But although the nerve can be directly stimulated by the galvanic current, yet this apparently does not occur when the semicircular canals are intact. The results of galvanic stimulation of the labyrinth do not agree with our experience in electrical stimulation of nerves in other parts of the body. The differences are the following:

The labyrinth can be stimulated by the galvanic current only, whereas a nerve can be stimulated by both galvanic and faradic currents. The stimulation of the labyrinth occurs during the flow of the current, whereas a nerve can only be stimulated during the make or break of the current. A change in the direction of the current causes a change in the direction of the nystagmus. This cannot be explained by assuming the nystagmus to be due to direct stimulation of the nerve.

The nystagmus lasts after the current has stopped, whereas the effect of electrical stimulation of a nerve does not continue after the current has stopped. The vestibular nerve is deeply situated in the skull, in close relationship with the cochlear and facial nerves. If the vestibular nerve were directly stimulated by the current, how can we explain the fact that neither the cochlear nor the facial nerve are stimulated at the same time as the vestibular nerve?

Breuer noticed that a much stronger current was needed to arouse nystagmus after the labyrinth was destroyed than before; and that when the labyrinth was destroyed both the anode and kathode caused movement of the head away from the stimulated side. This corresponds to nystagmus toward the stimulated side.

Barany explains the galvanic reaction in the following way:

There is a constant flow of impulses from both labyrinths to the eye-muscles. The impulses from the right labyrinth tend to cause nystagmus to the right, and those from the left labyrinth, nystagmus to the left. These impulses counterbalance each other, normally. When the kathode is applied to the ear the vestibular nerve is placed in a condition of kathelectrotonus, during which its conductivity is increased. As a result of this increase in conductivity, the impulses from the labyrinth of that side overbalance those from the opposite side, and the result is a nystagmus toward the side of the kathode. When the anode is applied to the ear the nerve is placed in a condition of anelectrotonus, during which its conductivity is diminished. This interferes with the transmission of the normal impulses

from the labyrinth of that side, so that they are overbalanced by the impulses from the opposite labyrinth. This results in a nystagmus away from the anode. This theory is controverted by the following fact; if cold water is poured into the right ear, there results a combined rotatory and horizontal nystagmus to the left. If the kathode is placed over the right ear at the same time, the kathelectrotonus of the right vestibular nerve ought to increase its conductivity for the caloric irritation, in which case the nystagmus toward the left should be increased. But, as a matter of fact, the galvanic nystagmus to the right neutralizes the caloric nystagmus to the left, so that the nystagmus ceases altogether.

Brünings believes he is able to diagnose a fistula in the external semicircular canal by means of the galvanic test. If, with the head in the optimum position for the external semicircular canal, the caloric reaction is diminished or absent, and there is a normal or increased galvanic excitability, he concludes that there is a fistula or a localized perilabyrinthitis in the external canal. He calls this the galvanic fistula test, and explains it as follows; the caloric reaction depends upon a free movement of the endolymph in the semicircular canals. However, the kataphoretic movement caused by the galvanic current is not interfered with by a stenosis of the semicircular canal, such as occurs with a circumscribed perilabyrinthitis. In fact, it may even be increased, on account of the improved conductivity for the current. Hence a diminished caloric reaction with a normal or increased galvanic excitability, means a mechanical interference with the flow of the endolymph current, and a normal nervous ampullary organ,

which is the condition in a fistula of the external semicircular canal.

THE FISTULA TEST

The fistula test can be elicited, as a rule, only in pathological conditions. The two factors which are necessary for its production are a fistulous opening in the bony labyrinthine wall, and functionating end-organs within the labyrinth. It cannot be elicited in the presence of a fistula, with a destroyed labyrinth.

The test consists in causing an endolymph-movement in the semicircular canals by means of compression and aspiration of the air in the external auditory canal and middle ear. This test is identical with *Ewald's* classical experiment on pigeons, with the pneumatic hammer. In *Ewald's* experiment, however, he was able to control accurately the direction of the endolymph current, whereas, in the fistula test, the factors which determine the direction of the endolymph current are not under our control.

Ewald exposed the semicircular canals of a pigeon, and made two small openings in the bony wall of the external canal, without injuring the membranous canal. Into the opening furthest from the ampulla he drove a metal plug, which completely obliterated the lumen of the canal at that point. Into the second opening, which lay between the plugged opening and the ampulla, he fitted a small pneumatic hammer, which he attached by means of plaster-of-Paris to the vertex of the pigeon's skull. The end of the hammer touched the membranous canal lightly. This hammer could be driven forward

and backward by compressing and relaxing a rubber bulb, which was attached to the hammer by means of a long rubber tube. As the canal was plugged at one point, the direction of the endolymph flow, upon compression and relaxation of the bulb, could be readily determined. On compressing the bulb the endolymph flow was toward the utricle, and on relaxation of the bulb it was away from the utricle (Fig. 59).

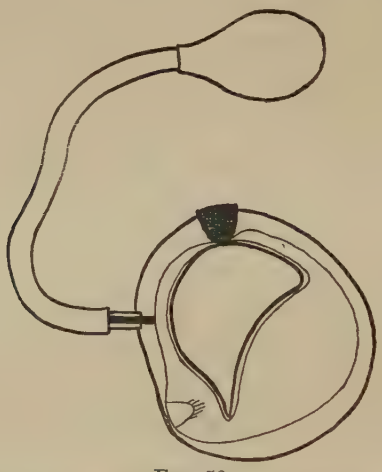

FIG. 59
DIAGRAM ILLUSTRATING EWALD'S EXPERIMENT ON PIGEON WITH PNEUMATIC HAMMER

Ewald observed that there was always a movement of the eyes and of the head in the direction of the endolymph flow, and in the plane of the canal which was stimulated. In the right external semicircular canal, on compressing the bulb, there was a rotation of the head and eyes to the left, in the horizontal plane, and on relaxation of the bulb the movement was to the right. With the left external canal the movements were reversed. Movements of the endolymph in the vertical

canals, he found, cause movements of the head and eyes in their planes also. In the external canals he found that the reaction resulting from an endolymph flow toward the utricle was greater than that resulting from an endolymph flow away from the utricle. In the vertical canals the opposite was the case; namely, the endolymph flow away from the utricle aroused a greater movement of the eyes and head than a flow toward the utricle. The movement of the eyes corresponds to the slow movement of the nystagmus, and the movement of the head corresponds to the reaction movement.

In the fistula test the direction of the endolymph movement depends on the location of the fistula, and the location of stenoses in the canals.

The fistula test is performed as follows: the olive tip of a Politzer bag is fitted air-tight into the canal of the ear. In cases where there is a mastoid wound, a Bier's cup is attached to the Politzer bag, and placed so as to cover the entire ear and wound. Slow steady pressure is made upon the bag. If a fistula is present, and the labyrinth is functionating, one of several things will happen. The most common result is a rotatory or horizontal or combined rotatory and horizontal nystagmus toward the examined ear. There may be a slow movement of the eyes away from the examined ear. Sometimes there is a nystagmus away from the examined ear, or a slow movement of the eyes toward the examined ear. Occasionally, accompanying the nystagmus, there is a movement of the head in the direction of the slow component of the nystagmus.

On releasing the bulb, and producing suction, there results a nystagmus or a slow movement of the eyes, in

a direction opposite to that produced by compression of the bulb. This nystagmus is usually weaker than that produced by compression, and it is often wanting altogether. Aspiration seems to produce a weaker stimulation than compression.

The commonest form of reaction to the fistula test, namely, nystagmus toward the examined ear, would seem to indicate that compression of the air in the external auditory canal and middle ear caused an endolymph flow in the external semicircular canal, toward the utricle, and aspiration, away from the utricle.

In performing the fistula test one must not use very much force, nor repeat the test too often, as it may cause a rupture of the membranous canal at the site of the fistula, with a spread of the infection from the middle ear into the labyrinth.

REACTION MOVEMENTS

Thus far we have concerned ourselves with only one of the reactions aroused by stimulation of the labyrinth, namely, nystagmus. There is a second reaction which can be aroused by stimulation of the labyrinth, that is, reaction movements of the extremities and trunk. Although these are not as important as nystagmus, for the purpose of diagnosis, yet a careful study of these movements will give us considerable additional information in regard to the condition of the labyrinth. Since the recent investigations by *Barany,* of the relationship between these movements and the cerebellar functions, they have proven of considerable importance in the diagnosis of cerebellar lesions.

The reaction movements are not a simple reflex like the nystagmic movements. They consist, according to *Barany,* of voluntary movements, which are modified in the cerebellar cortex by abnormal centripetal impulses from the semicircular canals. In the cerebellar cortex, motor impulses from the cerebrum are met by centripetal impulses from the semicircular canals. If these centripetal impulses are rendered abnormal, through disease or abnormal stimulation of the semicircular canals, the normal voluntary movements are changed in such a way as to result in reaction movements.

The reaction movements can be influenced to a certain extent by the will, and consequently they are most marked in individuals who have not a highly developed muscular and kinæsthetic sense, to warn them of the occurrence of the reaction movements. For this reason they are much more marked in children than in adults.

A reaction movement is a movement of the extremities or body, elicited by stimulation of the labyrinth, in the plane of the stimulated semicircular canal, and in the direction of the endolymph current, i. e., in a direction opposite to the nystagmus.

Barany was the first to notice the influence of the position of the head upon the reaction movements. The nystagmus remaining unchanged, a change in the position of the head will cause a change in the direction of the reaction movements.

We will first consider reaction movements of the extremities. If a patient is rotated to the left, a number of times, and then the rotation is stopped, there follows a horizontal after-nystagmus to the right. If, during

this nystagmus, the patient be told to extend his right arm straight in front of him, it will be seen that the arm slowly deviates to the left, i. e., in a direction opposite to the nystagmus. If the patient notices this deviation he will correct it by a rapid movement of the arm in the direction of the nystagmus.

If, with the horizontal nystagmus to the right, the head is tilted 90° to the left shoulder, the arm moves downward as the nystagmus is directed upward in space. If the head is tilted 90° to the right shoulder, the arm moves upward. If cold water is allowed to flow into the right ear, we have a rotatory nystagmus to the left. If the right arm is extended horizontally in the frontal plane it will deviate downward. The left arm will deviate upward. If the head is now tilted forward 90°, both arms will deviate to the right. If the head is tilted 90° backward, the arms will deviate to the left. If the head is turned 90° to the left, both arms held in front of the body will deviate downward. If the head is turned 90° to the right, both arms held in front of the body, will deviate upward. Provided the nystagmus and the position of the head remain unchanged, a change in the position of the arm will make no difference in the reaction movement. It makes no difference whether the arm is pronated or supinated, the movement will be to the left, provided the nystagmus is a horizontal nystagmus to the right, and the head is in the upright position.

The same reaction movements can be obtained with the legs, but these are not as constant as those in the arms.

In order to arouse reaction movements, much

stronger stimulation is necessary than to arouse nystagmus.

Barany has devised a method for testing reaction movements in the extremities, which he calls the pointing test. It can be applied to each joint separately, the shoulder, elbow or wrist, or the hip, knee or ankle.

The test (for the wrist-joint, for instance) is performed as follows: If spontaneous nystagmus is not present, nystagmus is aroused by rotation or cold water. With the eyes closed, the patient's forearm is allowed to rest on the back of a chair, and held in place with one of the examiner's hands.

The patient is told to perform dorsal flexion at the wrist, his index-finger being extended. The back of his index-finger is allowed to touch the palmar surface of the index-finger of the examiner's hand, which is held above that of the patient. The patient is now told to flex his hand as far as possible, and then to again touch the examiner's finger, which has not moved in the interval. If the patient has a horizontal nystagmus to the right, he will point past the examiner's finger to the left. It is advisable that the patient should not be warned of the fact that he has pointed past, as this will influence the succeeding tests. For this reason the examiner should touch the patient's finger, so as to make him believe that he has not pointed past.

As before mentioned, this reaction movement is aroused in the cerebellar cortex, as a result of impulses reaching the cerebellum from the semicircular canals and motor cortex. From the cerebellar cortex, probably from Purkinje's cells, the impulses reach the muscles of the extremities, through the brachium con-

junctivum, red nucleus and Monakow's tract. If a portion of the cerebellar cortex is destroyed, as the result of abscess, tumor, etc., irritation of the semicircular canals will fail to arouse a reaction-movement.

Barany was able to prove experimentally that there are certain areas in the cerebellar cortex which govern movements in certain joints, and in certain directions. In a case of cerebellar abscess of the right hemisphere, which had healed, and in which the cerebellum was covered only by cicatricial tissue, he froze portions of the cerebellar cortex by means of an ethyl-chloride spray, applied for about 3 minutes.

Before the freezing the patient pointed correctly with both arms and legs. On freezing the right cerebellar hemisphere, the patient pointed past to the right, with the right arm and leg. The left extremities were unaffected. On rotating him to the left, and producing a horizontal after-nystagmus to the right, the right arm and leg did not point past to the left. That is to say, there was no reaction movement. After two or three minutes the conditions again returned to the normal, i. e., there was no spontaneous pointing past, and on arousing nystagmus, there was a normal reaction movement.

On repeated examinations, by freezing small areas of the right cerebellar cortex, he came to the following conclusions:

1. During the freezing there is no nystagmus.

2. The left arm and leg are not influenced in their reaction movements.

3. Immediately behind the ear is the centre for movement to the left, of the right arm. When this spot is

frozen there occurs spontaneous pointing-past of the right arm to the right; and with horizontal nystagmus to the right, there is no reaction movement to the left.

4. Immediately behind the arm centre is the centre for movement to the left, of the right foot.

As the result of pathological findings, *Barany* believes that the centres for movement to the left, of the right elbow and wrist joints lie in front of the arm centre.

With a lesion in the fibre-tract between the cerebellar cortex and the anterior horn cells in the spinal cord, we also get a loss of the reaction movements of the extremities. But as the fibres are collected into a small bundle, a lesion here will cause a loss of reaction movements in numerous joints, in all directions, whereas a lesion in the cerebellar cortex will affect only one joint in one direction, unless the lesion is very extensive.

The reaction movements of the body are tested by having the patient stand up, with his feet together and his eyes closed. A reaction movement of the body is not noticeable if it occurs in the horizontal plane. If it occur in any other plane than the horizontal, however, it will cause the patient to fall. Just like the reaction movements of the extremities, so reaction movements of the body depend upon the canal which is stimulated, and the position of the head. In this way it is differentiated from the loss of equilibrium which is due to other causes, such as cerebellar disease or hysteria.

In the presence of a spontaneous nystagmus, the patient is told to stand up, with his feet together, and his eyes closed. If he has a rotatory nystagmus to the left, he will fall toward the right. If the head is now ro-

tated 90° to the left he will fall forward. If the head is turned 90° to the right he will fall backward.

If there is no spontaneous nystagmus present, the reaction movements of the body are best examined by means of the caloric test. (Rotation test with the head upright is of no use here, because it causes a reaction movement in the horizontal plane, which cannot be observed, as it does not cause the patient to fall.)

After eliciting a caloric nystagmus, the test is performed just as with spontaneous nystagmus.

A loss of reaction movements of the body in the presence of a powerful rotatory nystagmus means disease of the cerebellar worm, or of the tract which leads from the worm to the anterior horn cells of the cord, namely, through the nucleus tecti, tractus uncinatus and Deiterso-spinal tract.

The various methods of stimulating the labyrinth which we have described vary in their intensity. The caloric stimulation is the weakest of all. The stimulation by rotation is stronger, and stimulation by compression is the strongest; for there are many cases which do not respond to the caloric or rotation tests, and respond very readily to the fistula test.

Coming now to the second portion of our examination, namely, an examination of the spontaneous symptoms caused by disease of the labyrinth, we must distinguish between the manifest and the latent stages of the disease.

During the early or manifest stage of the disease the symptoms are usually very violent and easy to recognize. We examine for spontaneous nystagmus. In labyrinthine disease this is usually a combination of the

rotatory and horizontal form, and in a direction away from the diseased ear. We look to see whether it is present in every position of the eyes, or only in certain positions.

We notice the impairment of equilibrium. In severe cases the patient is unable to stand, always falling toward the diseased side. The direction of the fall is influenced by the position of the head, as described in the discussion of reaction movements.

The patient prefers to lie on the side of his sound ear. This is because of the fact that in lying down the tendency is to look away from the pillow. When the patient lies on the sound ear, on looking away from the pillow, the nystagmus, and consequently the subjective symptoms also, are diminished, for, as we have already mentioned, looking in the direction of the slow component diminishes the intensity of the nystagmus.

When questioned most patients complain of an apparent rotation of objects in the room. The direction of the apparent rotation is usually in that of the rapid component of the nystagmus, i. e., toward the sound ear. Sometimes objects appear to move in both directions, and rarely in the direction of the slow component of the nystagmus.

When the eyes are closed there is a sensation of rotation of the body in the direction of the rapid component of the nystagmus.

In many cases there is a history of nausea and vomiting.

In the late or latent stage of the disease we must look carefully for the spontaneous symptoms of the disease. These symptoms consist in impairment of equi-

librium. In this stage of the disease the impairment of equilibrium is so slight that special tests are required to bring them to light. Such a series of tests was elaborated by *von Stein.*

He divided his tests into two sets, the first set to determine the static muscular efficiency, and the second, the dynamic muscular efficiency.

The determination of the static muscular efficiency deals principally with the functions of the utricle and saccule. The tests are all performed with the eyes closed. They are as follows:

1. Standing still, with feet together.
2. Standing still, on toes, with feet together.
3. Standing on one foot.
4. Standing on an inclined plane.

In this fourth test a quantitative estimation is attempted. The plane on which the patient stands is gradually made more oblique until the patient falls. The instrument used is the goniometer. In a normal person, the anterior inclination which can be held without falling is 36° to 40°, the posterior inclination, 26° to 30°, and the lateral inclination, 37° to 38°. In disease of the labyrinth the angles are much smaller than these.

The determination of the dynamic muscular efficiency deals principally with the functions of the semicircular canals. These tests are also performed with the eyes closed. They are as follows:

1. Walking straight forward and backward on level floor.
2. Hopping on toes forward and backward, with feet together.

3. Hopping on one foot, forward and backward.

4. Rotation about vertical axis of body, with feet together, to right and left.

5. Rotation on one foot.

By means of these various tests small degrees of loss of equilibrium can be determined. However, the results of these tests are not absolutely to be relied upon, because other factors, besides the labyrinths, must be taken into account, namely, the muscular and joint senses. Impairment of these senses would cause the same loss of equilibrium as disease of the labyrinth. Besides, normal individuals show marked differences in regard to the ease with which they execute complicated manœuvres. Physical training during childhood has a good deal to do with this. However, as a supplement to our other methods of testing the functions of the labyrinth, these methods have their place.

Since the internal ear is composed of two parts, each of which has a separate and distinct function, it is necessary to extend our examination to the cochlea as well as to the vestibular apparatus.

In testing the hearing by the various means in vogue we wish to emphasize the fact that, particularly in the presence of unilateral labyrinthine disease, many of these tests are almost without value, unless the ear not to be tested is absolutely excluded from the possibility of hearing. In order to accomplish this, the closure of the external meatus with the moistened finger is not sufficient, and recourse must be had to more efficient means. Among the instruments designed for this purpose the noise apparatus of Barany and that of White are the best.

The usual means of testing the hearing in a suspected ear are the following:

1. Voice.
2. Whisper.
3. Acoumeter or watch.
4. Tuning forks.
    a. Weber.
    b. Rinné.
    c. Schwabach.

The **Weber test** is based upon the fact that any interference with the sound-conducting mechanism causes the tone of a vibrating tuning fork, placed upon the vertex, to be referred to the ear in which the interference exists. In the presence of unilateral disease of the sound perceiving apparatus, however, the tone is referred to the unaffected ear.

The **Rinné test** is a comparison between aerial conduction and bone conduction in the same ear. Normally, if a vibrating tuning fork is placed on the mastoid, and the perception of the tone has ceased, the fork should be heard again, if it be removed from the mastoid and held near the auricle, in front. This is called a positive Rinné. The time of perception of the tone by aerial conduction would be approximately twice that by bone conduction, provided the ear were normal. In this connection it must be remembered, that in the presence of unilateral labyrinthine disease, with the loss of cochlear function, the tone of a fork placed upon the mastoid of the diseased ear would be perceived through bone conduction in the sound ear. Under these circumstances the Rinné would be negative ∞. In other words, there would be no perception by aerial

conduction if the vibrating fork were held close to the diseased ear.

The **Schwabach test** consists of a comparison between the perception time, by bone conduction, of the suspected ear and that of an ear known to be normal. Thus the vibrating tuning fork is first placed upon the mastoid of the examiner. In this way the normal perception time is determined.

In examining an ear in which labyrinthine disease is suspected, the conclusions to be drawn from testing the hearing by means of the voice are decidedly uncertain, unless the unsuspected ear is absolutely excluded by the noise apparatus. If, after the application of such an instrument to the opposite ear, the individual who is examined fails to hear speech, no matter how loud, when emitted even close to the tested ear, we may safely conclude that the latter is deaf to the voice. In addition to testing with the voice, it has been our custom to test the suspected ear by whistling through the lips, the pitch of the note thus made approximating g^3. Here the blast of air must be directed in such a way that the patient is unable to feel it. So many individuals confuse the feeling of the blast of air in the whistling test and of the vibration of the forks in the tuning fork tests, with the hearing of the sounds, that the greatest care must be exercised lest this confusion lead to error. Needless to state that during the tests with the voice and by whistling, the patient must not observe the examiner.

Deafness to the voice, however, is not alone sufficent to enable us to conclude that all the cochlear function has been destroyed. To further substantiate this we

use the tuning fork tests. These alone may give us the data for a fairly certain diagnosis of total unilateral deafness. Thus, if the Weber is lateralized to the sound ear, while the Rinné is negative ∞, i. e., if the tuning fork is heard only by bone conduction, and if, in addition to this, the Schwabach shows a shortening of bone conduction for the high pitched forks while the low forks are not heard at all, then we have sufficient data to establish a highly probable diagnosis of unilateral deafness. It seems to us, however, that in the noise apparatus we have so sure a means of establishing a diagnosis of deafness positively and beyond a doubt, that we have come to rely upon this adjunct to our examinations.

The Weber test as usually made is notoriously uncertain. In combination with the noise apparatus, however, it gives us accurate and valuable information. This modified Weber test is carried out as follows; both ear pieces of the White apparatus are snugly fitted into the ears. The air stream is then turned on either from the balloon of the White instrument or from a compressed air apparatus. We now determine positively through voice, whistling and fork tests, that both ears are thus excluded from hearing. Then, after the air current is shut off, the ear piece in the suspected ear is removed, while that in the ear not to be tested is carefully held in place. The rubber tube connected to the ear piece which has been removed is clamped or knotted. After the air is again turned on the vibrating fork is placed upon the vertex, glabella, chin, teeth, or mastoid. If the patient hears the fork he can do so only with the tested ear. It is customary, in these tests, to

use a fork of medium pitch, c^2, 512 V., but the method is applicable to the high and low forks as well. It is in the tests with the low pitched forks, however, that the greatest confusion between the feeling of the vibration and the perception of the sound is encountered.

CHAPTER IV

PATHOLOGY

Labyrinthitis, or inflammation of the internal ear, may be classified in many different ways.

The most important method of classification, for practical purposes, is upon a clinical basis, into

1. Circumscribed labyrinthitis, and
2. Diffuse labyrinthitis.

The circumscribed form may be further subdivided into paralabyrinthitis, perilabyrinthitis, and a form in which there is a combined para- and perilabyrinthitis. Endolabyrinthitis alone has never been observed.

The diffuse form may be divided into a combined peri- and endolabyrinthitis, which is known as empyema of the labyrinth, and panlabyrinthitis, which is a combination of para-, peri- and endolabyrinthitis.

Paralabyrinthitis is an inflammation of the bony capsule of the labyrinth. Perilabyrinthitis is an inflammation of the perilymph spaces, and endolabyrinthitis is an inflammation of the endolymph spaces.

Both the circumscribed and the diffuse forms of labyrinthitis may be acute, subacute or chronic.

The above outlined clinical basis of classification is important, because upon it are based, to a large extent, our indications for treatment.

A second method of classification is based upon the pathologic changes in the labyrinth. According to this classification, we divide labyrinthitis into the exudative, plastic and necrosing forms.

PLATE IX.

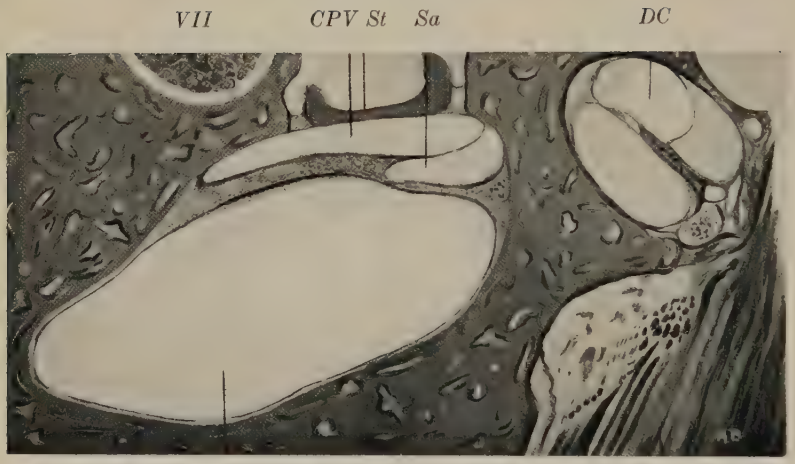

Fig. 60

Diffuse Serous Labyrinthitis

DC—Dilated ductus cochlearis
U—Dilated utricle
Sa—Saccule
St—Foot-plate of stapes
CPV—Compressed cisterna perilymphatica vestibuli
VII—Facial nerve
IAC—Internal auditory canal

PLATE X.

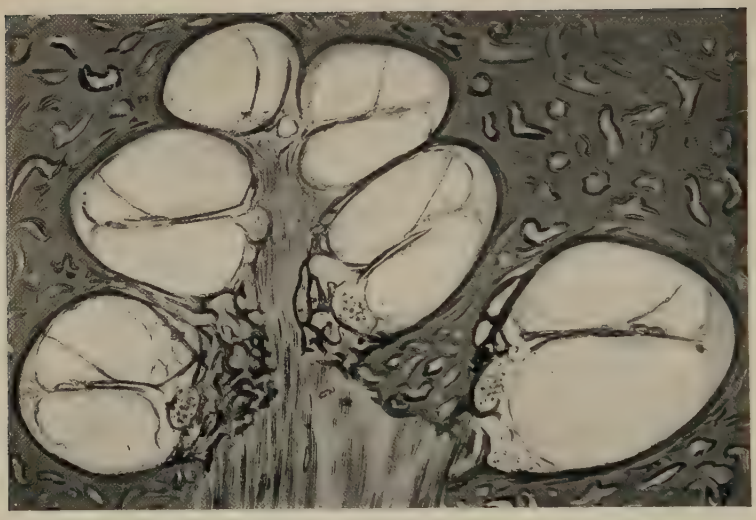

Fig. 61

Diffuse Sero-Fibrinous Labyrinthitis

Deposits of fibrin in the peri- and endolymphatic spaces of the cochlea

The exudative may be subdivided into the serous (Fig. 60, Plate IX) and purulent types; the plastic may be subdivided into the sero-fibrinous (Fig. 61, Plate X) and fibrino-purulent forms; and the necrosing may be divided into those in which the necrosis is limited to the soft tissues, and those in which the bony capsule is invaded also.

Etiology forms the basis for a third method of classification. Here we have the scarlatinal, cholesteatomatous, tuberculous, traumatic forms, etc.

Finally, we may classify labyrinthritis upon an anatomo-topographic basis, i. e., according to the location of the portal of infection. According to this method we may divide the cases into primary and secondary. No undoubted case of primary labyrinthitis has ever been reported. The secondary cases may be divided into meningeal, tympanic and metastatic types.

In the meningeal cases the infection reaches the labyrinth in one or more of the following ways:

1. Through the internal auditory canal.
2. Through the aqueductus cochleæ.
3. Through the aqueductus vestibuli.
4. By necrosis of the inner labyrinthine wall.
5. Through the hiatus subarcuatus.

In the tympanic cases, the infection reaches the labyrinth through one or several of the following pathways:

1. Oval window.
2. Round window.
3. Fistula in one of the semicircular canals.
4. Fistula in the promontory.

The metastatic cases are those in which the infection

reaches the labyrinth through the circulation, as in mumps. These cases are very rare.

The most common location for circumscribed labyrinthitis is in one of the semicircular canals. Here the conditions are most favorable for circumscription of an inflammatory process. The gradual onset of the infective process, the small calibre of the bony canals, and the large number of connective tissue septa in the perilymph spaces, favor a walling off of the inflammatory focus from the rest of the labyrinth. However, a circumscribed labyrinthitis may occur in almost any portion of the labyrinth. A low-grade infection through the oval window may give rise to a localized inflammatory focus in the cisterna perilymphatica of the vestibule. *Alexander* states that the thick fibrous septum which separates the cisterna perilymphatica from the utricle is firm enough to resist the spread of infection for a considerable length of time. This septum divides the labyrinth into two portions. On one side lie the utricle and semicircular canals, and on the other the cisterna perilymphatica, the saccule and the cochlea (Fig. 12, Plate VIII). An inflammatory focus can remain localized to one or the other side of this septum for some time.

The perilymph spaces of the ampullæ are almost completely shut off from the vestibule by connective tissue septa. These serve to limit an inflammatory process within the perilymph spaces of the semicircular canals, or in the cisterna perilymphatica of the vestibule. With inflammatory thickening of the cristæ, these septa may form an effective barrier across the entire lumen of the bony canals.

The inflammatory process may involve the entire perilymph space, leaving the endolymph spaces free.

In inflammation of the cochlea it is possible for the disease to be limited to the first half of the basal whorl. In fact, this is the most common location for a circumscribed inflammatory focus in the cochlea. Even in the cases of diffuse labyrinthitis the inflammatory changes are more advanced in the first half of the basal whorl than in the rest of the cochlea. The inflammation seems to start in the beginning of the basal whorl.

Ruttin explains these facts upon a purely mechanical basis. In cases where the middle ear infection is not very virulent, a perforation through the annular ligament of the stapes, which develops gradually, results first in a deposit of pus on the vestibular surface of the foot-plate. After a sufficient quantity has accumulated a portion of the pus falls down into the beginning of the scala vestibuli of the basal whorl of the cochlea, and rests on Reissner's membrane. The lower half of the basal whorl forms the lowermost portion of the cochleal cavity in every position of the head. *Ruttin* found that by placing a triton-shell, with its axis in a position corresponding to the human modiolus, and removing a portion of the outer wall of the upper half of the basal turn, a globule of mercury dropped through this opening into the lower half of the basal turn could not be made to leave this portion of the canal, when the position of the horn was changed to correspond to various positions of the human head. From this experiment he draws the conclusion that the reason for the frequent circumscription of an inflammatory process in

the lower half of the basal whorl of the cochlea is a mechanical one.

When a perforation occurs through the secondary tympanic membrane the infective process reaches the beginning of the scala tympani first (Fig. 62, Plate XI). If the infection is mild the inflammatory process is very likely to be limited to the scala tympani of the lower half of the basal whorl of the cochlea.

Consequently, with the same intensity of infection, the prognosis with regard to function is worse in perforations through the oval window than in those through the round window. For in the former both the vestibule and the cochlea are invaded, while in the latter only the cochlea is involved.

Ruttin states that where a purulent involvement of the cisterna perilymphatica results in a rupture into the endolymph spaces, the location of the rupture is always in a deeply situated portion of the membranous labyrinth, i. e., in the saccule, canalis reuniens, coecum vestibulare, or the vestibular portion of the ductus cochlearis.

The most common situation for a circumscribed labyrinthitis is in the external semicircular canal. The most common cause is cholesteatoma. A portion of the bony capsule of the external semicircular canal lies exposed in the aditus. In this situation it is subjected to the eroding action of the cholesteatoma. As the bony capsule is eroded it is replaced by granulation tissue. When the destructive process has gone through the entire thickness of the bony capsule, the endosteum, covered on its outer surface by granulations, is exposed at the bottom of the fistulous opening. Up to this stage

PLATE XI.

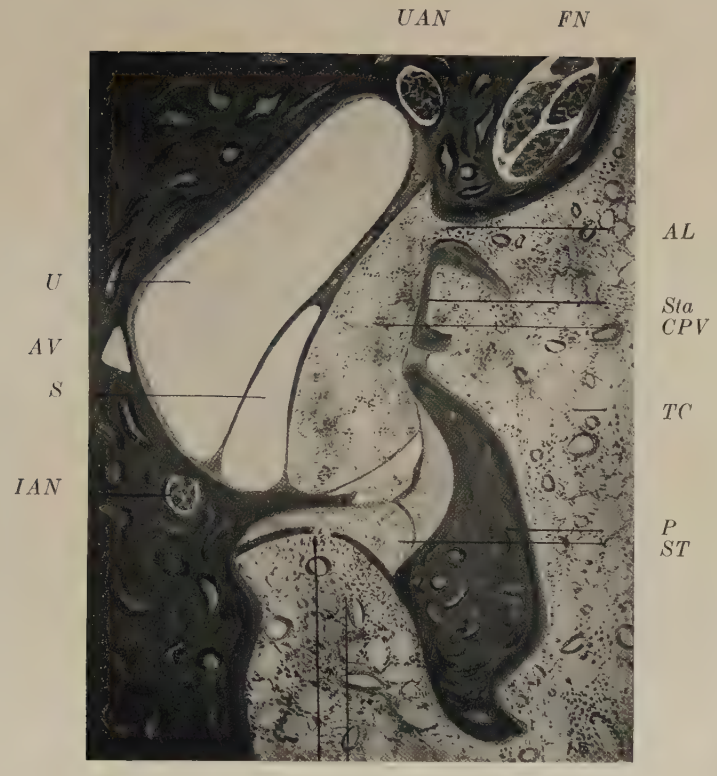

FIG. 62

CHRONIC PURULENT OTITIS MEDIA, WITH PERFORATION THROUGH THE UPPER PART OF ANNULAR LIGAMENT OF STAPES, AND THROUGH SECONDARY TYMPANIC MEMBRANE

AL—Perforation through upper part of annular ligament
STM—Perforation through secondary tympanic membrane
CPV—Purulent exudate in cisterna perilymphatica vestibuli
ST—Purulent exudate in scala tympani of beginning of cochlea
TC—Tympanic cavity filled with pus and granulations
P—Promontory
RRW—Recess of round window
U—Utricle (not involved)
S—Saccule (not involved)
AV—Beginning of aqueductus vestibuli
Sta—Stapes
FN—Facial nerve
UAN—Utriculo-ampullaric nerve
IAN—Inferior ampullaric nerve

PLATE XII.

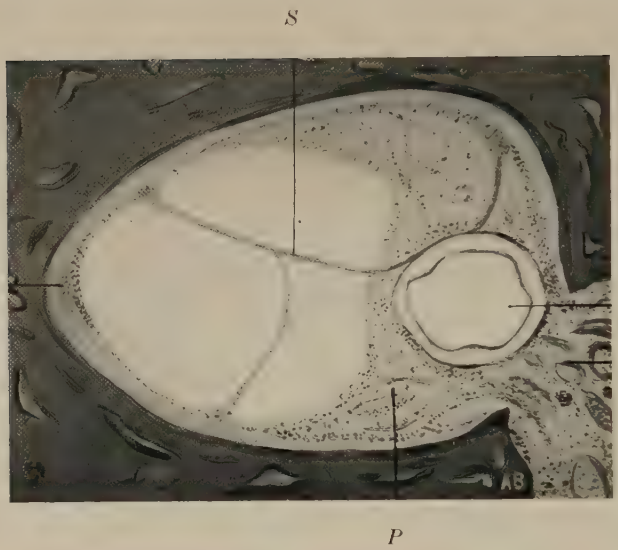

FIG. 63

FISTULA IN EXTERNAL SEMICIRCULAR CANAL

Paralabyrinthitis. Suppurative perilabyrinthitis. Membranous canal is normal

F—Fistula in bony capsule filled with granulation tissue. Endosteum is destroyed in this location

M—Normal membranous canal

P—Granulation tissue and pus in perilymph space

E—Endosteum

S—Connective tissue septum in perilymph space

the process is known as a paralabyrinthitis. It may remain in this stage for a considerable length of time. Sooner or later, however, the granulations erode through the endosteum and enter the perilymph spaces. In response to the irritation of the infective process there results an inflammatory thickening of the endosteum and of the connective tissue septa, which bridge across the spaces between the endosteum and the membranous semicircular canal. This thickening shuts in the inflammatory focus and prevents the infection from becoming general. The condition is now called a perilabyrinthitis (Fig. 63, Plate XII). The wall of the membranous canal and its contents are normal.

After a time the wall of the membranous labyrinth becomes hyperæmic and thickened, and there occurs a fibrinous deposit upon its inner surface. The crista becomes infiltrated with round cells. Its epithelial elements become swollen and necrotic. Small abscesses may form in the wall of the membranous labyrinth. This wall finally perforates and the endolymph space becomes filled with pus. The infection in the endolymph space may remain limited to the one semicircular canal or it may spread through the entire labyrinth.

While fistulæ are most common in the external semicircular canal, they may occur in any of the canals, or, in fact, in any portion of the labyrinthine capsule. When they occur in the superior or posterior canal they are much more apt to escape observation at the time of operation, on account of the spongy character of the bone which surrounds these canals.

The fistula which is commonly seen in the wall of the external semicircular canal is usually trough-shaped.

It is about 1mm wide. It averages 3 to 4 mm in length. Sometimes granulations are seen in the canal. It is extremely rare for pus to be seen exuding from the fistula. In some cases the entire eminence of the canal is destroyed, and there are seen two holes, each corresponding to one limb of the canal.

When the bony wall has been partially eroded, either from within or from without, the contents of the semicircular canal can sometimes be seen, as a dark brown streak, through the very thin layer of bone that remains. This is sometimes mistaken for a fistula, but such a case would naturally give a negative fistula test.

A diffuse labyrinthitis may be diffuse from the beginning, or it may start as a circumscribed labyrinthitis and become diffuse by breaking down its barriers.

Cases of diffuse labyrinthitis may change to the circumscribed form after a time. On microscopic examination these cases show a circumscribed labyrinthitis, with degenerative changes in the nerve end-organs of the remaining portions of the labyrinth.

The cases of diffuse labyrinthitis may be divided into serous, sero-fibrinous, and purulent types. The serous and sero-fibrinous types may be classed together, as there is no way of differentiating them clinically, and their course and outcome is the same. The purulent cases may be divided into those with and those without bone involvement. The cases of purulent labyrinthitis without bone involvement are known as empyema of the labyrinth. When the bony capsule is involved the condition is known as a panlabyrinthitis.

Diffuse serous labyrinthitis may be either secondary to a circumscribed purulent labyrinthitis, with a fistula,

or it may spread through an intact labyrinthine wall. The latter form is called induced serous labyrinthitis.

Induced serous labyrinthitis may occur during the course of an acute or chronic purulent otitis media. It may come on after a radical mastoid operation, either within 24 to 48 hours after the operation, or at any later time, until the cavity has healed.

It was at one time thought that induced labyrinthitis was due to a transmission of the infection from the middle ear to the labyrinth through the minute vascular anastomosis which exists in the outer labyrinthine wall, between the tympanic and the labyrinthine circulations. But this is probably not true; for if it were so, induced labyrinthitis would be a much more common condition than it is. *Voss* considers it to be a collateral inflammatory œdema of the labyrinth, resulting from the middle ear inflammation.

O. Mayer, Lange and *Nager* examined cases histologically, which have demonstrated the true pathogenesis of this condition. In *Mayer's* case there was an acute purulent inflammation of the middle ear. The recesses of the oval and round windows contained large numbers of bacteria. The annular ligament of the stapes and the secondary tympanic membrane showed an œdema, with a beginning necrosis of the cellular elements of these membranes. On the vestibular surfaces of the foot-plate of the stapes and the secondary tympanic membrane, as well as in the various other portions of the labyrinth, there were fibrinous deposits. There were a few round cells in the fibrinous deposits, which had apparently migrated from the dilated vessels in the ligamentum spirale. There were no bacteria

to be found either in the peri- or the endolymphatic spaces of the labyrinth, nor in the annular ligament or secondary tympanic membrane.

The sequence of events was probably as follows: the bacteria in the middle ear, which had accumulated in the recesses of the oval and round windows, on account of the favorable conditions for their development in these locations, elaborated toxines, which passed into the annular ligament and secondary tympanic membrane, and then through these structures, into the perilymph and endolymph spaces of the labyrinth. Here the irritation of these substances produced a dilatation of the labyrinthine blood-vessels, with an exudation of serum, fibrin, and a few round cells. *Mayer* believes that labyrinthitis toxica would be a better name for the condition than induced labyrinthitis.

If the necrotic changes in the annular ligament and secondary tympanic membrane are sufficiently intense, perforation takes place, with the occurrence of a purulent labyrinthitis.

Just as a purulent inflammation of the middle ear may cause an induced serous labyrinthitis, so a purulent peri labyrinthitis may give rise to a serous inflammation in the endolymph spaces. We sometimes see in histological examinations purulent inflammation in one portion of the labyrinth and serous inflammation in another portion.

The pathological changes which occur in a diffuse serous or sero-fibrinous labyrinthitis consist of a dilatation of the labyrinthine blood-vessels, an increase in the peri- and endolymphatic fluids, deposits of fibrin in various portions of the labyrinth, migration of a few

round cells into the peri- and endolymphatic spaces, swelling and some necrosis of the epithelial elements in the nerve end-organs, and desquamation of endothelial cells.

Increase in the perilymph is shown by dilatation of the cisterna perilymphatica vestibuli, with compression of the utricle and saccule, and compression of the ductus cochlearis. Increase in the endolymph is shown by dilatation of the utricle and saccule, and especially by ectasia of the ductus cochlearis (Fig. 60, Plate IX). The ductus cochlearis may be three or four times its natural size. Reissner's membrane is bowed out with its convexity toward the scala vestibuli, and its point of attachment to the outer cochleal wall is displaced upward.

Ruttin explains the occurrence of ectasia of the ductus cochlearis in several ways.

He noticed that in many cases the ectasia was limited to the basal whorl of the cochlea, while the ductus cochlearis was collapsed in the upper whorls. He believes that in some of the cases the increase of the perilymph in the upper whorls causes a collapse in the ductus cochlearis. The fluid which is forced out of this portion of the endolymph tube causes a dilatation of the ductus cochlearis in the basal whorl.

In other cases an increase of the endolymph causes a dilatation of the ductus cochlearis directly.

Ruttin noticed that as far as the ectasia extended, Reissner's membrane was covered by a layer of fibrinous exudate on the side toward the scala vestibuli. He believes that this fibrinous exudate falls by reason of its weight from the cisterna perilymphatica vestibuli

into the scala vestibuli of the lower half of the basal whorl of the cochlea. The thickening which is thus produced suffices to hold Reissner's membrane in the abnormal position, into which it has been pushed by the increase of endolymph in the ductus cochlearis.

He believes that in some cases the fibrinous deposit, in the process of organization, causes traction on Reissner's membrane, producing in this way an ectasia of the ductus cochlearis.

Diffuse serous or sero-fibrinous labyrinthitis is always acute. It never lasts more than a week or two, and results in complete restitutio ad integrum, or in permanent destruction of portions of the labyrinth with secondary atrophic changes.

A purulent infection which reaches the labyrinth from the middle ear almost always makes its entrance through a rupture in the labyrinthine wall. But it can occur as the result of emigration of bacteria through an unruptured annular ligament, as in a case observed by *Grünberg,* or through an unruptured secondary tympanic membrane, as in a case observed by *O. Mayer.*

Diffuse purulent labyrinthitis may be acute or chronic. In the acute cases bone destruction is uncommon, except in scarlatinal infections. In the chronic cases bone destruction is very frequent.

The pathological changes which occur in diffuse purulent labyrinthitis are divided by *Alexander* into mechanical lesions and inflammatory changes. The mechanical lesions are due to the breaking through of the pus, and consist of the tearing of Reissner's membrane and of other portions of the membranous labyrinthine

wall, mechanical destruction of the neuro-epithelial cells, and displacement of the membrana tectoria.

The inflammatory changes consist of dilatation of the blood vessels, infiltration of the soft tissues with serum and round cells, exudation of pus into the perilymph and endolymph spaces, and swelling and necrosis of the epithelial cells. The hairs are more resistant than the bodies of the hair-cells. The hairs of the cristæ and maculæ are more resistant than those of Corti's organ.

The neuro-epithelium and nerve-fibres may remain intact for some time after the endolymph spaces are filled with pus. A case examined by *Alexander,* in which the labyrinthine functions could not be aroused before death, showed, on post-mortem examination, the labyrinth filled with purulent exudate, but the nerve end-organs intact. From this he draws the conclusion that a normally constituted endolymph fluid is necessary to enable the end-organs to functionate properly.

After a time the end-organs are completely destroyed. Suppuration may extend into the aqueductus cochleæ and aqueductus vestibuli. But the entrances to these passageways are often occluded by a swelling of their fibrous or epithelial lining or by desquamated epithelial cells. In this way a spread of the infection through these canals is prevented.

The spiral ganglion is usually infiltrated with round cells. The nerve-fibres in the modiolus and in the internal auditory canal, as well as the lymph spaces about the nerve-fibres, are infiltrated with round cells. The nerve-fibres may be degenerated.

Politzer and *Lange* found in many cases an abscess

at the fundus of the internal auditory canal, over the areæ cribrosæ, which was sharply differentiated by a well-marked line of circumscription from the remainder of the nerve-bundle. This abscess lies in the subarachnoid space, and is walled off by the attachment of the arachnoid sheath of the nerves to the dural lining of the internal auditory canal, which occurs near the fundus of the canal, and which separates the subarachnoid from the subdural space in this situation.

An abscess located at the fundus of the internal auditory canal destroys not only the nerve fibres, but also the blood vessels which supply the soft tissues and portions of the bony capsule of the labyrinth, namely, the internal auditory artery. The interruption of their blood-supply results in necrosis of these tissues. Necrosis may also be caused by the inflammatory process within the labyrinth causing embolism or thrombosis of the branches of the internal auditory artery.

The endosteum is the most resistant of all the soft structures within the labyrinth, and reacts to the inflammatory irritation by becoming thickened, and by the formation of granulations upon its free surface. These granulations may partly or completely fill the labyrinthine spaces, and may cause destruction of their bony walls (Fig. 64, Plate XIII).

The destructive bone changes which occur in diffuse purulent labyrinthitis are of two kinds, caries and necrosis. Usually both forms are present together.

In caries of the labyrinthine capsule the inflammation usually starts in the membranous labyrinth. When the inflammatory process in the membranous labyrinth is very intense there occurs necrosis of the soft tissues.

PLATE XIII.

FIG. 64

CHRONIC PURULENT LABYRINTHITIS, WITH CARIES OF BONY WALL OF SEMICIRCULAR CANAL FROM WITHIN

SC—Perilymph space of semicircular canal, full of granulation tissue and connective tissue

MC—Remnant of membranous canal full of pus

C—Inner portion of bony wall of canal destroyed by carious process

PLATE XIV.

OW *St* *TC* *P* *C* *C* *C*

V *IAC* *A*

FIG. 65

DIFFUSE PURULENT LABYRINTHITIS FOLLOWING LARGE PERFORATION IN REGION OF OVAL WINDOW

OW—Large perforation in region of oval window
St—Remains of stapes
TC—Tympanic cavity filled with pus and granulations
V—Vestibule, with membranous portion destroyed
P—Promontory
C—Cochlea, filled with purulent exudate
A—Sharply circumscribed abscess in fundus of internal auditory canal
IAC—Internal auditory canal, containing cochlea and vestibular nerves infiltrated with pus cells

This arouses a reactive inflammation in the surrounding bony capsule with its periosteal covering. Granulations invade the bone canals, and cause a resorption of the bone tissue, the destroyed bone being replaced by granulation tissue. Caries is an interstitial granulation involvement of bone. The Haversian canals become wider and wider. The dense bone becomes porous and the cavities are filled with round cells, a gelatinous intercellular substance and numerous blood-vessels. Osteoclasts lie in Howship's lacunæ. The bone corpuscles lie in partly opened bone canals.

This inflamed bone has a strong affinity for eosin stain. At a later stage the bone takes very little stain or none at all.

The picture is one of bone resorption. There is a softening, perhaps through pathological decalcification, during which the cells, which before lay in bone-canals, become free, swell up, and change their shape.

Necrosis of the labyrinthine capsule consists of death of the bone, due to a cutting off of its blood-supply. A large portion of the labyrinthine capsule receives its blood-supply through the internal auditory artery. This vessel is practically an end-artery, as the anastomosis between it and the middle-ear vessels, through the promontory, is so minute as to be negligible. An abscess in the internal auditory canal (Fig. 65, Plate XIV), by destroying the internal auditory artery, will cause necrosis of a large portion of the labyrinthine capsule. In acute scarlatinal middle-ear disease the infective process is so intense that it causes necrosis of the soft tissues within the labyrinth. This results in destruction of the branches of the internal auditory

artery in the labyrinth, and thus portions of the bony capsule are rendered necrotic.

As a result of necrosis of a portion or all of the bony capsule there is set up a reactive inflammation in the surrounding bone in the form of caries. The surrounding bone is gradually eroded until eventually the necrotic bone lies free, as a sequestrum, in a bed of granulation-tissue. As the necrotic bone contains no blood-vessels it cannot be absorbed. If a portion of the inner tympanic wall is included in the necrosis the sequestrum may be extruded through the middle ear. If no portion of the tympanic wall is included in the necrosis the sequestrum lies enclosed in a bony shell, and cannot be extruded until the overlying bone is destroyed by carious inflammation.

The cochlea is the portion of the labyrinth which is most frequently extruded as a sequestrum (Fig. 66, Plate XV). Necrosis of the semicircular canals or vestibule is far less common. Exfoliation of only a portion of the labyrinth does not mean that the disease is limited to that portion. Usually the entire labyrinth is diseased.

No case of undoubted primary labyrinthitis has yet been observed. Labyrinthitis may be secondary to intracranial disease, to disease in the middle ear, or it may be metastatic. Of intracranial diseases, by far the most common cause of labyrinthitis is epidemic cerebro-spinal meningitis. According to various statistics from 15 to 50% of the cases of cerebro-spinal meningitis are followed by deafness. In 19 temporal bones taken from 10 patients who died of epidemic cerebro-spinal meningitis, *Goerke* found inflammatory changes

PLATE XV.

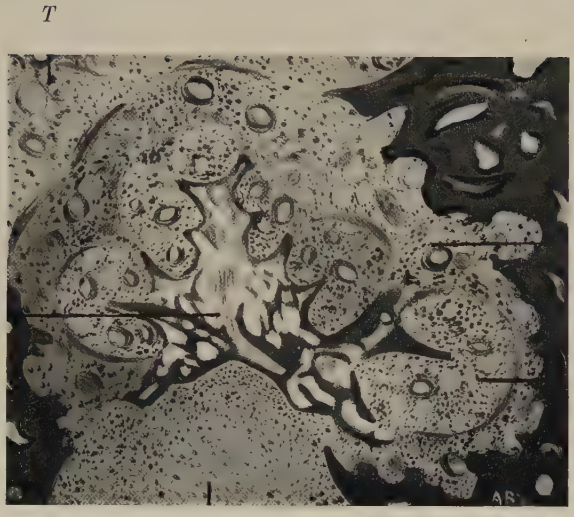

FIG. 66

CHRONIC DIFFUSE PURULENT LABYRINTHITIS, WITH CARIES OF LABYRINTHINE CAPSULE AND NECROSIS AND SEQUESTRATION OF MODIOLUS

M—Necrotic modiolus
IAM—Abscess at bottom of internal auditory canal
B—Cochleal canal full of granulations
C—Carious labyrinthine capsule replaced by granulation tissue
T—Tympanic cavity full of granulation tissue

in the internal ear 17 times. In most of the cases there was a circumscribed labyrinthitis present.

According to *Friedrich* and *Hinsberg,* there is 1 case of labyrinthitis in every 100 cases of middle-ear suppuration. At first glance these figures would seem to indicate that intracranial disease was a much more prolific cause of labyrinthitis than is disease of the middle ear. But middle ear disease is so much more common than cerebro-spinal meningitis that the number of cases of labyrinthitis actually caused by the former is much greater than that caused by the latter.

In addition to cerebro-spinal meningitis, labyrinthitis can be caused by purulent leptomeningitis, or by epidural abscess in the posterior or middle fossa. A labyrinthitis which is caused by a middle ear infection may spread to the meninges. The meningitis may reinfect the labyrinth or infect the labyrinth of the opposite ear through the internal auditory canal or aqueductus cochleæ.

The most frequent pathways of infection from the intracranial cavity to the labyrinth are through the internal auditory canal and through the aqueductus cochleæ. Infection through the aqueductus vestibuli is rare, but it does occur. A subdural abscess may erode the bony capsule of the posterior or superior semicircular canal, and thus infect the labyrinthine cavity.

Hinsberg observed a case in which an extradural abscess, caused by infection through the vessels which pass through the fossa subarcuata, eroded the wall of the superior semicircular canal, and thus infected the labyrinth.

The cases of labyrinthitis which are secondary to middle ear conditions, we may divide into those due to inflammatory disease of the middle ear, and those due to injuries.

The middle ear inflammations which cause labyrinthitis may be acute or chronic. Chronic middle ear disease is responsible for the vast majority of the cases. Out of 137 cases of labyrinthitis observed by *Jansen* only three occurred with acute otitis media. But in the chronic middle ear conditions an acute exacerbation of the inflammation is usually responsible for the extension into the labyrinth.

The most common of the acute middle ear diseases which cause labyrinthitis is that due to scarlatina. Scarlatinal otitis is especially apt to cause bony destructive changes in the labyrinth.

Of the chronic middle ear conditions cholesteatoma is responsible for most of the labyrinthine infections. Next in frequency is tuberculosis of the middle ear. Of 121 cases of fistulæ of the semicircular canals observed by *Jansen,* 71 occurred with cholesteatoma and 16 with tuberculosis.

Cholesteatoma causes labyrinthine disease by producing a gradual erosion of the bony labyrinthine capsule, to which is added an infective process from the middle ear cavity. The cholesteatoma sometimes extends into the labyrinth.

Middle ear suppuration may extend to the labyrinth through a fistulous opening in one of the semicircular canals, through the oval window, the round window, or through a fistulous opening in the promontory. *Goerke* reported a case where extension to the cupola

of the cochlea occurred through a fistulous opening at the tympanic orifice of the Eustachian tube. *Grunert* reported a case where the infection took place through a fistulous opening in the facial canal.

In regard to the relative frequency of the point of entry of the infection there is still some dispute. It is probable that the most frequent point is through a fistula in the external semicircular canal. The statistics which are compiled from clinical observations indicate an overwhelming predominance of fistulæ in the semicircular canals, whereas histological examinations show a predominance of fistulæ in the oval window. The reason for this is that infections which take place through the oval window are much more apt to produce fatal complications than those occurring through a fistula in the semicircular canal.

The oval and round windows are favorable sites for the transmission of disease to the labyrinth, on account of the fact that the recesses of these windows fill with granulations and connective tissue, causing a stagnation of pus in these regions. The annular ligament and the secondary tympanic membrane, being membranous, offer less resistance than the bony capsule, to the destructive process. The various changes which may be seen in the oval window are:

1. Destruction of part or the whole of the annular ligament.
2. Destruction of part or the whole of the foot-plate of the stapes.
3. Displacement of the foot-plate of the stapes.
4. Destruction of the cartilaginous and bony margin of the oval window.

5. Pus may be seen exuding from the oval window, or it may be filled with granulations.

Defects in the secondary tympanic membrane cannot be observed clinically, on account of the position of this membrane in the wall of the recess of the round window.

There may be one or several fistulæ leading from the middle ear into the labyrinth. When two or more fistulæ are present they may all be primary; i. e., they may all arise from the disease-process in the middle ear, and act as portals of entry for the infection into the labyrinth; or one fistula may be primary and the remaining fistulæ secondary to the disease within the labyrinth. Out of 14 cases of labyrinthitis examined microscopically by *Lange,* he found a single primary fistula in 3 cases, multiple primary fistulæ in 6 cases, and primary and secondary fistulæ in 5 cases. The primary fistulæ were located in one of the semicircular canals 9 times, in one of the windows 4 times, and in the windows and semicircular canals once.

Lange explains the occurrence of secondary fistulæ as follows; he found that secondary fistulæ occurred only where there was necrosis of the soft tissues within the labyrinth. The labyrinthine contents become infected, for example, through a fistula in the external semicircular canal. If a necrosis of the soft tissues in the labyrinth occurs, there is set up a reactive inflammation in the surrounding tissues. As the oval and round windows lie at the border line between the necrotic and reactive tissues, the annular ligament and the secondary tympanic membrane are among the first tissues to be destroyed in the demarcation zone. In this way sec-

ondary fistulæ occur. The secondary fistula can occur in any portion of the labyrinthine wall.

Lange does not believe the term fistula should be applied to the primary cases, but only to the secondary cases, as, strictly speaking, a fistula means an opening through which an abscess cavity has established communication with the surface, and indicates that the disease has progressed from within outward.

The traumatic cases of labyrinthitis may be divided into those due to fracture at the base of the skull, those due to hemorrhage, those due to a projectile or foreign body, and those caused by operation.

In fracture through the base of the skull the line of fracture often passes through the internal ear. The eighth nerve may be torn through in the internal auditory canal. Even if the line of fracture does not pass through the internal ear the functions of the labyrinth may be destroyed as the result of a hemorrhage into its cavity. Bullets or foreign bodies, such as hatpins, or a paracentesis knife may enter the labyrinth through the external auditory canal and middle ear.

Operation, especially a radical mastoid operation, may be a causative factor in producing a labyrinthitis, in several ways:

1. By causing dislocation of the stapes.
2. By opening the external semicircular canal or promontory.
3. The traumatism of the operation may result in the breaking down of adhesions about a circumscribed labyrinthitis, making it diffuse. This has been determined histologically in several cases. There was found an old

fistula, with evidences of recent inflammation in the remainder of the labyrinth.

4. The trauma may increase the virulence of inoffensive bacteria which are present in the middle ear.

The infection is occasionally carried to the labyrinth through the circulation, as in mumps. But this means of transmission is comparatively rare.

A labyrinthitis may end in one of three ways:

1. Restitutio ad integrum.
2. Healing with permanent changes.
3. Extension to the intracranial cavity.

Complete return to the normal is possible only in the serous and sero-fibrinous forms. But every case of serous and sero-fibrinous labyrinthitis does not return to the normal. In a certain number, as well as in a large number of the purulent cases, atrophic changes occur. These atrophic changes are seen in the maculæ, cristæ, and organ of Corti, in the ganglion spirale, and in the nerve-filaments leading to the end-organs (Fig. 67, Plate XVI).

Wittmaack describes three grades of degenerative changes in the organ of Corti.

1. *Mild degree.*—Degeneration of a few sensory epithelial cells, with normal supporting cells.

2. *Moderate degree.*—Degeneration of many sensory epithelial cells, with moderate flattening of the supporting structures, especially of Deiters' cells. The rods are somewhat depressed. There is adhesion of the tectorial membrane to the organ of Corti.

3. *Marked degree.*—The organ of Corti consists of a flat epithelial hillock, without recognizable sensory or

PLATE XVI.

FIG. 67

COMPLETE ATROPHY OF CORTI'S ORGAN AND SPIRAL GANGLION

CO—Atrophic organ of Corti *SG*—Atrophic spiral ganglion

PLATE XVII.

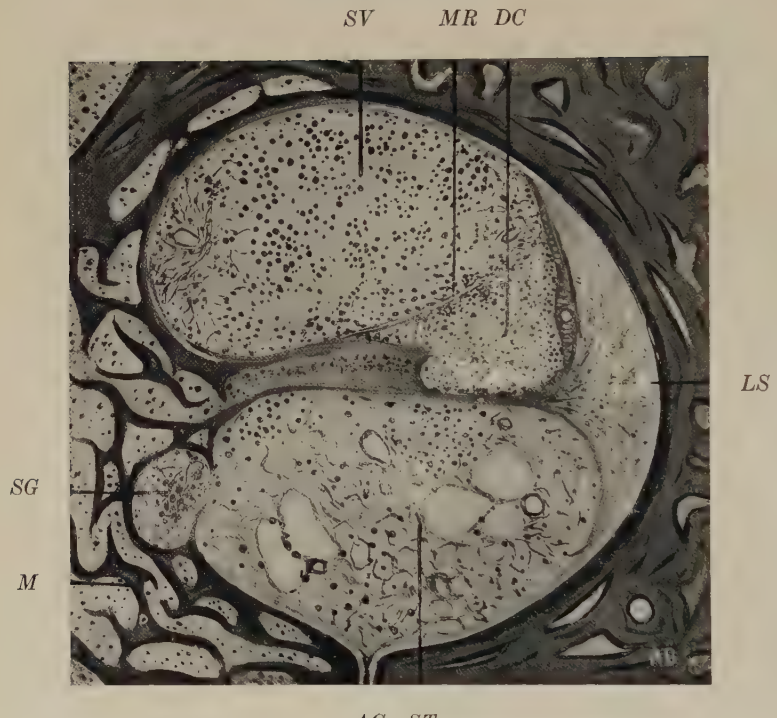

FIG. 68

DIFFUSE SUPPURATIVE LABYRINTHITIS WITH BEGINNING ORGANIZATION

SV—Scala vestibuli of cochlea, full of pus. Beginning organization at medial and lateral margins

MR—Reissner's membrane

DC—Ductus cochlearis, full of pus. Organ of Corti and membrana tectoria are partly destroyed

ST—Scala tympani filled with connective tissue

AC—Cochleal opening of aqueductus cochleæ

LS—Ligamentum spirale infiltrated with round cells

SG—Spiral ganglion infiltrated with round cells

M—Modiolus

PLATE XVIII.

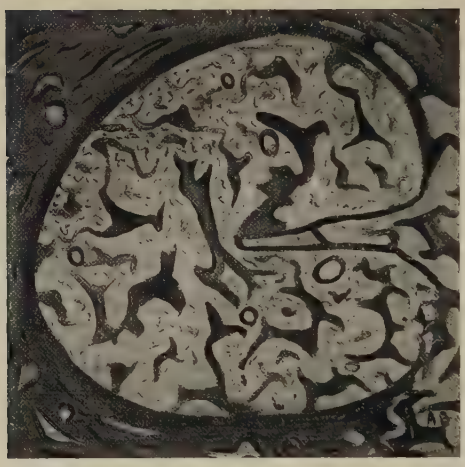

Fig. 69

Chronic Suppurative Labyrinthitis, Which Has Healed, the Purulent Exudate Becoming Organized; the Cochleal Cavity is Entirely Filled With New-Formed Bone, and Connective Tissue

supporting cells, to the surface of which the membrana tectoria may be adherent.

In the ganglion spirale there is shrinking of the protoplasm of the ganglion nerve cells. Vacuoles appear. There is disappearance of the Nissl granules, and finally complete destruction of the cells. The canal of Rosenthal is then occupied by an empty network of connective tissue.

In a large proportion of the cases of purulent labyrinthitis, organization of the exudate takes place. The round cells are replaced by connective tissue cells, which are gradually transformed into connective tissue fibres (Fig. 68, Plate XVII). Thus the entire labyrinthine cavity may be filled with connective tissue.

The irritation of the endosteum gives rise to the production of a hyperplastic or ossifying periostitis, which results in a bony transformation of the connective tissue, which fills the labyrinthine cavity (Fig. 69, Plate XVIII). The new-formed bone is distinguished from the older bone by

1. Its greater affinity for eosin.
2. The lack of a regular lamellar structure.
3. The presence of irregular large bone corpuscles.
4. The simultaneous occurrence of all stages, from osteoid tissue to finished bone.

The infection may spread from the labyrinth to the intracranial cavity. It may result in meningitis, cerebellar abscess, epidural abscess, interdural abscess, or sinus thrombosis. The most frequent of these complications is diffuse purulent leptomeningitis. Circumscribed purulent leptomeningitis and the serous form

also occur. *Alexander* states that more than 80% of the cases of otitic cerebellar abscess are due to labyrinthitis.

The pathways of infection from the labyrinth to the intracranial cavity are:

1. The internal auditory canal.
2. The aqueductus cochleæ.
3. The aqueductus vestibuli.
4. Along the vessels which pass through the fossa subarcuata.
5. Through necrosis of the posterior or superior surfaces of the petrous pyramid.
6. By metastasis.

The most frequent pathways are the internal auditory canal and the aqueductus cochleæ. Extension through these channels usually results in a diffuse purulent leptomeningitis. Extension through the other pathways is more apt to result in the production of a localized intracranial lesion.

Fortunately in many cases the lining membrane of the aqueductus cochleæ reacts to the irritation of the inflammatory process within the labyrinth in such a way that the lumen of the canal is closed by swelling of the soft tissues. Otherwise every case of diffuse purulent labyrinthitis must of necessity spread to the meninges, as this passageway ends in the subarachnoid space by means of a wide open mouth. Furthermore, its inner opening is situated in the floor of the beginning of the scala tympani of the basal whorl of the cochlea, where the cochlear inflammation is most often localized.

In the internal auditory canal the infection travels

through the foramina in the areæ cribrosæ, and along the sheaths of the nerve-fibres.

Politzer found the following changes in the internal auditory canal in cases in which this canal served as the avenue of infection to the intracranial contents.

1. Pus along the nerve-fibres.
2. Pus in the empty nerve-canals in the areæ cribrosæ.
3. Pus between the nerves and the inner walls of the meatus.
4. The walls of the meatus partly destroyed by caries.
5. An abscess in the fundus of the canal.
6. The modiolus partly or wholly destroyed.
7. Nerve fibres infiltrated with pus cells, or with hemorrhagic extravasations.
8. Nerve fibres replaced by connective tissue. The connective tissue enters the meatus from the interior of the labyrinth.
9. In several cases there was a sharp line of demarcation between the peripheral infiltrated portion of the nerve and the central portion, which was more or less normal.

In most cases, with purulent infiltration in the internal auditory canal, the facial nerve was intact. This is explained by *Lange* by the fact that the facial nerve has greater resisting power than the eighth nerve; by the fact of its arachnoidal sheath being more closely adherent to it; and by the fact that it is supplied by a separate vessel, which accompanies it from the middle ear (a branch of the stylo-mastoid artery).

Steinbrügge, Habermann, Herzog and *Alexander* de-

scribed cases in which there was inflammatory infiltration of, and hemorrhage into the facial nerve, without any impairment of its function.

Infection sometimes reaches the intracranial cavity through the aqueductus vestibuli. This is not a very common pathway, for the reason that the lining-membrane of the passageway often becomes so swollen as to occlude its lumen. Where the infection does travel along this route it results in an empyema of the saccus endolymphaticus. This is a small sac lying between two layers of the dura on the posterior surface of the petrous bone, about half way between the internal auditory meatus and the inner margin of the lateral sinus.

An empyema of the saccus endolymphaticus is very apt to spread beyond the limits of the sac, between the two layers of the dura which enclose it, especially in an outward and downward direction, where the meshes of the fibrous tissue are very loose. Here the abscess is apt to lie in the posterior wall of the lateral sinus, the lateral sinus lying to its outer side, and the cerebellum to its inner side (Fig. 70, Plate XIX). *Kramm* reported a case in which such an interdural abscess ruptured in both directions, resulting in an infective sinus thrombosis and cerebellar abscess.

However, every collection of pus in the location of the saccus endolymphaticus is not necessarily a saccus-empyema. A histological examination is necessary to determine this point. It is necessary to demonstrate the fact that the walls of the abscess cavity are lined by a layer of epithelial cells, in order to be certain that we are dealing with a case of saccus-empyema.

PLATE XIX.

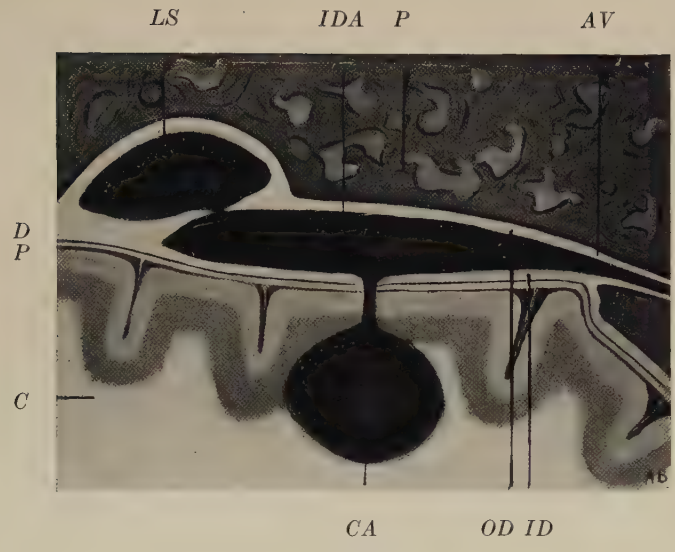

FIG. 70

EMPYEMA OF SACCUS ENDOLYMPHATICUS RESULTING IN INTERDURAL ABSCESS, SINUS THROMBOSIS AND CERELELLAR ABSCESS

P—Posterior surface of petrous bone
AV—Outer aperture of aqueductus vestibuli
IDA—Interdural abscess resulting from empyema of saccus endolymphaticus
LS—Lateral sinus
D—Dura mater
OD and *ID*—Outer and inner layers of dura enclosing saccus and interdural abscess
P—Pia mater
C—Cerebellum
CA—Cerebellar abscess

The following conditions may exist in the saccus region, according to *Lange:*

1. Interdural abscess without involvement of the saccus. Such a case was described by *Wagener.*

2. *Lange* saw a case of interdural abscess below the apertura externa of the aqueductus vestibuli, resulting from destruction of the bony labyrinthine capsule at this point.

3. Empyema of the saccus, extending through the ductus endolymphaticus from a labyrinthine suppuration. Such cases were described by *Goerke* and *Kramm.*

4. Infection of the saccus endolymphaticus from an epidural abscess was observed by *Lange.*

5. Infection of the saccus from a thrombosed lateral sinus was observed by *Lange.*

6. Empyema of the saccus endolymphaticus may perforate through its outer wall into the epidural space, giving rise to an epidural abscess.

In cases of necrosis of the soft tissues within the labyrinth, there is always necrosis of the ductus endolymphaticus up to the apertura externa of the aqueductus vestibuli, where the ductus joins the saccus endolymphaticus. The saccus is never involved, because it has a separate blood-supply. It is supplied by the dural vessels.

The vessels which pass through the fossa subarcuata run from the spongiosa in the medial antral wall, under the arch of the superior semicircular canal, to the dura of the posterior fossa. They are surrounded by a process of the dura. With disease of the bone about the superior semicircular canal the infection may spread to

these vessels, and result in thrombosis of the inferior petrosal sinus, or epidural abscess.

Suppuration in the middle ear or mastoid cells may give rise to an epidural abscess on the superior or posterior surfaces of the petrous bone. Pus in these locations can erode the bony capsule of the superior or posterior semicircular canal, and invade their contents.

Suppuration in the labyrinth may give rise to infective thrombi in the labyrinthine vessels. In this way the intracranial contents may be infected by metastasis.

CHAPTER V

SYMPTOMS OF LABYRINTHITIS

The inner ear combines two organs whose functions are absolutely distinct from each other. The cochlea is concerned only with hearing, while the static labyrinth (vestibule and semicircular canals) is one of the peripheral sources of those impulses whose function it is to establish our equilibrium. As in every other end-organ, so in the internal ear as well, there are definite functional disturbances if the nerves which supply the end-organs are either irritated or destroyed.

In the cochlea nerve irritation makes itself known through subjective noises, while nerve destruction results in deafness. Here the differentiation is clear and well defined. In the static labyrinth, on the other hand, the signs of nerve irritation are so similar to those of nerve destruction, that no positive differentiation can be made and no hard and fast line can be drawn between them.

If a static labyrinth is abnormally stimulated, so that its impulses prevail over the other impulses for the preservation of body balance, there follow functional disturbances with a nystagmus directed to the side of the stimulated labyrinth. These phenomena we have called **signs of stimulation disharmony.** If a static labyrinth is destroyed, then there ensue functional disturbances, essentially the same as those which follow abnormal stimulation, but the nystagmus is di-

rected to the sound side. To these disturbances we have applied the term **signs of destruction disharmony.**

As the functions of the two organs in the inner ear differ, so too do the symptoms that result from their irritation and destruction by disease. If the cochlea is affected there arise subjective noises and loss of hearing, while disease of the static labyrinth causes vertigo, disturbances in equilibrium, nausea, vomiting and nystagmus.

Subjective noises are very inconstant. While they may occur at any stage of the labyrinthitis, they are but rarely sufficiently annoying to arouse complaint. Undoubtedly the reason for this lies chiefly in the fact that the other disturbances are vastly more important to the patient, so that the subjective noises are crowded out of his consciousness. Occasionally, too, subjective noises have existed for some time, and the patient does not differentiate between those which were caused by the chronic purulent otitis media and those due to the labyrinth infection. Subjective noises may occur in the presence of a diffuse labyrinthine suppuration, when hearing has been totally destroyed. It has been shown that, in cases of diffuse suppurative labyrinthitis, cells in localized areas of Corti's organ may remain unaffected by the pathological changes. If one may judge from the histological appearance of these cells, it is probable that they are still susceptible to irritation. The subjective noises, therefore, may be due to stimulation of these intact cells by the inflammatory products in the cochlea. The noises may persist, or even begin only after the operative destruction of the

labyrinth. *Neumann* believes this to be due to degenerative changes in the ganglion cells.

So long as a labyrinthitis is localized the hearing, as a rule, is not completely lost. In some cases, indeed, it is surprisingly good. As soon, however, as the pathological process becomes diffuse, either spontaneously or as the result of a radical mastoid operation, hearing is abolished. There are, of course, exceptions to this rule. In the beginning of even a diffuse purulent labyrinthitis there may be some hearing, but it is retained for only a very short time.

In acute diffuse labyrinthine suppuration the hearing is usually lost before the functions of the static labyrinth are destroyed. This is probably due to the fact that the hair-cells of the organ of Corti are less resistant than those of the maculæ and cristæ. In cases of diffuse serous labyrinthitis the hearing is frequently completely lost, and may remain so even after the labyrinthine inflammation has subsided.

Vertigo and disturbances of equilibrium show the widest variations in their intensity. While in some cases there may be only a slight sense of unsteadiness, in others the disturbances may be so severe that the patient is unable to walk unsupported. The more rapid the destruction of the end-organs within the labyrinth, the more violent are the disturbances in balance.

Nausea and vomiting commonly accompany the other disturbances that result from labyrinthine disease. Vomiting usually follows active movements, but it is sometimes aroused by passive movements as well. Lifting a patient in or out of bed may bring on an attack. The vomiting due to a labyrinthitis differs

from that caused by intra-cranial complications in that the former is always accompanied by nausea and is never of the projectile type. Food, per se, does not arouse the vomiting in labyrinthitis so much as the movements of the head incident to taking nourishment.

In addition to the symptoms described and the nystagmus, which we have reserved for separate consideration, there are two of considerable importance, viz., fever and facial paralysis.

From many observations it has been established that an uncomplicated labyrinthitis, no matter of what type, causes little or no elevation of temperature. This is not surprising when we consider that from this minute closed bony cavity little or no absorption can take place. Therefore any considerable rise of temperature associated with a labyrinthitis must be regarded as an indication that the disease is probably extending beyond the confines of the labyrinth. If such a rise of temperature cannot be ascribed to other causes, and if the functional tests of the labyrinth indicate destruction of the end-organs, radical interference is the only means at our command of averting a meningeal infection.

The inclusion of a portion of the facial nerve in the labyrinthine capsule, renders it extremely liable to be involved when the labyrinth is attacked by disease and its walls are destroyed. *Neumann* states that 80% of cases of labyrinthine necrosis are accompanied by facial paralysis. From the rapidity with which, in some instances, these paralyses clear up after the radical mastoid operation has been performed, we must conclude that they are often due not to a neuritis, but to the pressure of sequestra or of granulations.

Just as in the phenomena which result from artificial stimulation of the normal end-organ, so too in the spontaneous disturbances that are caused by labyrinthine disease, nystagmus is in the foreground of clinical interest. The spontaneous nystagmus shows the same components (i. e., slow and rapid) as that which is aroused artificially. With regard to its plane, it resembles closest the nystagmus which is aroused through the action of the galvanic current. As a rule a nystagmus caused by disease within the labyrinth, is of a combined rotatory and horizontal type. As an explanation for this, the theory has been offered that in disease processes in the labyrinth the three cristæ are irritated equally. While this might hold good so long as there were signs of stimulation disharmony, i. e., so long as the impulse aroused by irritation was transmitted to the centres, it certainly fails to explain why the nystagmus of destruction disharmony also has a combined rotatory and horizontal character.

With regard to the direction of the nystagmus, a majority of cases of labyrinthitis follow the rule that the nystagmus is directed to the sound side.

Like the artificial, the spontaneous nystagmus is influenced by the direction in which the eyes are turned. Frequently a spontaneous nystagmus becomes evident only when the eyes are turned to the side toward which the rapid component is directed. Increasing the resistance to the nystagmic movements by turning the eyes toward the side of the slow component, retards and may even completely abolish the nystagmus. It has been shown above how this fact is made use of in estimating the intensity of the nystagmus, as well as in

making the functional tests when a spontaneous nystagmus is present.

Just as in the artificially aroused phenomena, so in the spontaneous disturbances resulting from labyrinthine disease, the nystagmus is accompanied by subjective and objective symptoms. We find here the familiar sensation of turning of the body, and the apparent turning or passing by of objects in the environment. These follow the same laws which govern the analogous reactions to artificial stimuli. Thus they are directed similarly with regard to the direction of the nystagmus (rapid component), and are intensified or retarded by corresponding fixation of the eyes.

With the spontaneous nystagmus, too, there are linked the reaction movements. From a diagnostic point of view these are of greatest importance. In the presence of a vestibular nystagmus, the reaction movements of the body are so constituted that they cause a tendency to fall in the direction of the slow component of the nystagmus. Thus if a nystagmus to the left exists, the body will have a tendency to fall to the right. If the face be turned toward the left shoulder, the body will fall forwards, and if the face be turned to the right shoulder the body will fall backwards. We have, therefore, in this test a means of determining whether the disturbances of equilibrium are of vestibular origin or not. If the tendency to fall is not in the direction of the slow component, whatever the position of the head, we must believe that the disturbances are not of vestibular origin.

The severity of the symptoms of labyrinthitis is proportional to the intensity of the disease process, and

particularly to the rapidity with which the latter spreads over the nerve endings. The remarkable variations which are observed in the symptoms resulting from labyrinthine disease, can be explained by differences in the pathological processes. The more rapidly the function of the organ is destroyed, the more apoplectiform and severe are the resulting symptoms. On the other hand, in the insidiously destructive processes, the symptoms may be so slight that they give rise to no disturbances of which the individual is conscious.

In the earlier descriptions of this affection, the symptoms which accompanied a labyrinthitis were considered to be the result of irritation in the diseased organ. But as our knowledge of labyrinthine functions widened, and particularly through the medium of the modern functional tests of the static labyrinth, this opinion became untenable, and has now been almost universally abandoned.

The symptoms of labyrinthine disease are identical with the phenomena which occur after the application of cocaine to the membranous labyrinth, or after the destruction of this organ or the eighth nerve. This fact leaves no doubt that they are due, in most instances, to the abolition of the normal function of the organ.

The fact that the symptoms of labyrinthine disease so closely resemble the phenomena following artificial stimulation of the end-organs of the static labyrinth, is not so remarkable when we consider that both are due to disturbances in the normal harmony of all the centripetal impulses, which have for their purpose the preservation of our body balance.

A disturbance of equilibrium which has its origin in

the labyrinth, can arise in but one of the following two ways. First, it can be due to an overbalancing by the impulses from an abnormally aroused labyrinth over the normal static influences from the other peripheral sources (stimulation disharmony), or, secondly, it may be due to a loss of the normal influences of the static labyrinth. This would cause an overbalancing by the remaining normal centripetal impulses (destruction disharmony). In the first instance (stimulation disharmony), the resulting reaction would be influenced directly by the over-stimulated end-organ. In the second instance (destruction disharmony), the reaction would be influenced only indirectly by the end-organ whose function was abolished, and would be influenced directly by the impulses from the remaining centripetal balance sources. Herein, according to *Wittmaack,* lies the explanation of the fact that the nystagmus resulting from stimulation disharmony changes its plane, according to the canal in which the impulse arises, while, on the other hand, the nystagmus due to destruction disharmony always has a uniform combined rotatory and horizontal character.

It was formerly believed that the phenomena, which resulted from the destruction of a labyrinth, were due to an overbalancing by the impulses from the remaining sound side. This view, however, does not agree with the observations after experimentally destroying first one and then the second labyrinth in the same animal.

If we destroy one labyrinth completely, or cut through one eighth nerve, there follow typical signs of destruction disharmony, i. e., disturbances of equilibrium with nystagmus directed to the sound side. So long as the

animal cannot orient itself without the help of impulses from the destroyed labyrinth, the disturbances of balance will continue. If, when they have disappeared, and the animal is again apparently normal, we destroy the second labyrinth or cut through the remaining eighth nerve, there will ensue disturbances of equilibrium quite as severe as those which followed the first operation. Now, however, the nystagmus is directed to the side of the first-destroyed labyrinth, despite the fact that this has remained totally without function.

Thus it is apparent that these disturbances depend neither exclusively nor even chiefly upon the impulses from the labyrinth itself. There can be no doubt that the manifestations, after the destruction of a labyrinth, result from an exceedingly complicated process, in which all the remaining centripetal balance impulses play a part. How the latter share in this activity cannot be determined, but it is clear that the result is largely influenced by vestibular function, even though the vestibular end-organs are, in this regard, secondary in importance to the vestibular centres.

The fact that no nystagmus occurs when both labyrinths are destroyed simultaneously indicates that the disharmonic impulses, aroused through the simultaneous destruction of both end-organs, oppose each other completely, in the same way that the impulses from both labyrinths aroused by simultaneously applied caloric or galvanic irritation neutralize each other. If, however, even in the presence of totally destroyed end-organs, a pathological process (meningitis, abscess, tumor, etc.) should attack the retrovestibular paths, typical symptoms of balance disturbance may develop.

Barany believes that the disturbances in equilibrium, which, together with deafness, follow the labyrinthitis that complicates cerebro-spinal meningitis, are due to the involvement of the cerebellar cortex, and not to the destruction of the labyrinths. With this view *Voss* also agrees.

Early in a pathological process in the labyrinth the nerve endings are irritated and signs of irritation or stimulation disharmony occur. Later, when the nerves are destroyed by the disease and the organ has lost its power to functionate, signs of destruction disharmony supervene. It must not be supposed, however, that at any stage of its development a pathological process in the labyrinth can arouse impulses through physiological means, i. e., through endolymph movements.

Pathological processes in the labyrinth destroy the normal function of this organ in several ways. The normal activity of the end-organs may be disturbed by changes in the endolabyrinthine pressure or by changes in the physical properties of the endolabyrinthine fluids. Again, inflammatory changes may attack the end-organ itself and destroy its power to functionate, and, lastly, changes in the nerve fibres and ganglia may prevent the transmission of impulses to the centres.

As we have stated above, it is but reasonable to suppose that, during the inception of a disease process within the labyrinth, the end-organ is irritated, and, therefore, there should occur signs of stimulation disharmony. It seems that these actually occur, even though in man it is rare to observe them. There have been observed, in early labyrinthine inflammations, evidences of stimulation disharmony with a nystag-

mus directed to the diseased side. On the other hand, cases have been recorded in which, despite the fact that the diseased labyrinth was still irritable, there was a pronounced nystagmus to the sound side. Here is an apparent contradiction which, up to the present, has not been satisfactorily explained. Sometimes signs of stimulation disharmony (nystagmus to the diseased side) coexist with those of destruction disharmony (nystagmus to the sound side).

An attempt to explain this contradiction has been made by assuming that, coincident with the disease in the end-organ, there occur changes in the nerve which reduce the conductivity of the latter. Just as in disease of the cochlear nerve, subjective noises and loss of hearing occur simultaneously.

Against this assumption, however, must be urged the fact that the nerve is apparently very resistant, and even after complete destruction of the labyrinth retains its irritability for a long time. For several months after a labyrinth is destroyed in an animal, nystagmus may be aroused by the application of the galvanic current. It must not be forgotten, however, that there is a vast difference between the effect that aseptic destruction of the labyrinth has upon the nerve and that caused by destruction of this organ by disease. Moreover, it has been shown that even in early serous labyrinthitis advanced changes in the nerve endings, fibres and even ganglia of both the cochlear and vestibular nerves can occur. On the other hand, *Ruttin* found a positive reaction to the galvanic test in cases in which the labyrinth operation had been performed two or three years previous.

It is remarkable, nevertheless, that in observing cases of labyrinthitis, even in their earliest stages, we so rarely see signs of irritation, i. e., stimulation disharmony. *Wittmaack* states that the impulses aroused by stimulation disharmony oppose those aroused by destruction disharmony, and it is not remarkable if the latter, because of their greater strength, completely overshadow the former. According to this idea there would be a constant struggle for supremacy, between the impulses of stimulation disharmony and those of destruction disharmony, and since, in the development of the disease, the factors that augment the impulses of destruction disharmony at the same time weaken those of stimulation disharmony the former soon prevail. Thus in labyrinthine disease a nystagmus directed solely to the diseased side is rarely observed.

If the impulses of stimulation and destruction disharmony are about equally strong, there is no nystagmus so long as the eyes are directed forward.

But if, under these conditions, the eyes be turned to one side or the other, a nystagmus directed to the side toward which the eyes are turned will develop. By turning the eyes to one side, we increase the resistance to that nystagmus which is directed opposite to the side to which the eyes are turned, and at the same time diminish the resistance to the nystagmus which is directed to the side toward which the eyes are turned.

A nystagmus directed to both sides, if it be of vestibular origin, occurs as a rule only when the function of the diseased labyrinth has not been totally destroyed, i. e., the labyrinth is still irritable. We find it, therefore, as we should expect, in cases of circumscribed

labyrinthitis. In these cases the diseased labyrinth responds to the functional tests. As soon as the end-organ is destroyed, or its function is interfered with beyond a point where it is capable of being irritated or of transmitting irritation, signs of destruction disharmony, with nystagmus directed to the sound side, supervene.

A vestibular nystagmus, therefore, which is directed to both sides must be regarded as evidence of incomplete destruction of function in the diseased labyrinth. *Alexander* strongly supports this view by the statement that this combination of nystagmus, both to the sound and to the diseased sides, occurs at the beginning of labyrinthitis, and when, after having been abolished, the functions return as the inflammation subsides.

A consideration of the symptoms of labyrinthitis is incomplete without a word regarding the functional tests of the static labyrinth. Unfortunately these tests create powerful impulses, out of all proportion to those which are aroused through the normal activity of this organ. The tests themselves differ in the strength of the impulses which they arouse. Thus the weakest among them is the caloric test. The response to this may be lost and the labyrinth still be susceptible to rotation and to the fistula test. Again, the response to the fistula test may be retained alone, while neither the caloric test nor rotation is capable of stimulating the diseased labyrinth.

The simultaneous application of galvanic or caloric stimulation to both sides gives us a means of comparing the susceptibility of the nerves and the end-organs of

the two sides. However, the ratio between the loss of irritability and the loss of function has not yet been determined. We have seen an instance of labyrinthine fistula in which there was a marked response to all the functional tests. At the time of the radical mastoid operation, the labyrinth was destroyed mechanically and thereafter remained totally without function. If this labyrinth had been functionating normally, as the responses to the tests led us to suppose, its sudden destruction should have entailed severe symptoms of destruction disharmony. On the contrary, those that ensued were exceedingly mild. We must conclude, then, that here was an instance in which, despite the fact that the labyrinth responded readily to all the functional tests, its functional activity had decreased and the centres had already accommodated themselves to the partial loss of the normal impulses.

All cases of chronic purulent otitis media, and in particular those cases in which the radical mastoid operation is contemplated, should be thoroughly examined as to function. It is true that the functional tests of the static labyrinth offer little in the way of accurate knowledge regarding the quantity of function retained, but the condition of the labyrinth, determined by its susceptibility to the functional tests, reveals accurately, as a rule, the pathological changes that have taken place within this organ. The estimation of these changes is of greatest importance, for upon it rests, in large measure, our determination of the surgical indications.

From a clinical standpoint labyrinthitis may be divided into serous and suppurative, circumscribed and

diffuse, latent and manifest. These divisions are but stages of the same pathological process. In reality the clinical picture of serous (and sero-fibrinous) labyrinthitis is neither a clear nor a sharply defined one. It is probable that a serous labyrinthitis, like a serous pleuritis, is in many instances but a forerunner of a purulent process. Whether and when the serous exudate becomes purulent depends upon the intensity and duration of the inflammatory process. In fact, both serous and suppurative forms may co-exist in the same labyrinth.

Although these categories are more or less artificial, they are, nevertheless, of great value; first, because they furnish us with definite clinical groups, and, secondly, because the surgical indications are determined, for the most part, strictly in accordance with the clinical data that form the basis for these groups.

A **circumscribed labyrinthitis,** as a rule, gives rise to attacks of vertigo, disturbances of equilibrium, nausea and vomiting; in short, to those phenomena which we call labyrinthine symptoms. Their onset is sudden and, after the first attack, there are frequent recurrences which sooner or later lead these patients to seek relief. In many instances, where the attacks are of short duration, they are undoubtedly due more to circulatory disturbances and changes in the intralabyrinthine pressure brought about by defects in the bony walls, than to pathological changes within the labyrinth itself. In these cases the vertiginous attacks are frequently brought on by stooping, straining or moving the head rapidly backwards and forwards or from side to side.

There is more or less loss of hearing, yet the hearing, in many of these cases of circumscribed labyrinthitis, is remarkably good, and it must be borne in mind that good hearing does not preclude the existence of a circumscribed labyrinthitis. Subjective noises are infrequent. In 50 cases reported by *Ruttin* they were present in only 17, severe in but 2. As a rule there is a history of only occasional tinnitus.

At the time of the examination the vertiginous attack may or may not be present, i. e., the labyrinthitis may be manifest or latent. If these symptoms are not present they may frequently be aroused by the rapid movements of the head described above.

There may be no spontaneous nystagmus, or there may be a rotatory nystagmus directed either to the diseased side or to the sound side, or to both sides, according to the direction in which the eyes are turned.

Rotation shows both labyrinths to be functionating normally, the caloric reaction and the fistula test are positive.

Diffuse serous labyrinthitis may follow a circumscribed labyrinthitis spontaneously or after a radical mastoid operation. It may occur, too, where there has been no circumscribed labyrinthitis and where the labyrinthine capsule is intact. This induced labyrinthitis probably results from the absorption, by the labyrinth, of the toxic products of bacterial activity in the cavities adjacent to the inner ear. If a diffuse serous labyrinthitis follows the radical mastoid operation, it manifests itself from the first to the third day, i. e., from 12 to 72 hours after the operation. In contradistinction to this, the signs of destruction disharmony,

which ensue after the labyrinth is injured mechanically during a radical mastoid operation, appear at once.

When a diffuse serous labyrinthitis has run its course, and either restitutio ad integrum or destruction of function has occurred, the disease cannot be considered latent, so that only the manifest form comes into consideration.

In a diffuse serous labyrinthitis there is a rapid diminution in hearing associated with labyrinthine symptoms, i. e., nystagmus directed to the sound side, nausea and vomiting, vertigo and disturbances in balance, which, if the patient be in bed, manifest themselves in a disposition to lie on the sound side, so that the eyes may be readily directed to the side of the slow component of the nystagmus.

The severity and duration of the symptoms vary in different instances. *Ruttin* states that, on an average, they last from 3 to 5 days. There are cases of circumscribed labyrinthitis in which, on the day following the radical mastoid operation, there ensue labyrinthine symptoms (i. e., loss of hearing, vertigo, nausea and vomiting and nystagmus) of such short duration that they must be ascribed to circulatory disturbances, or probably to pressure from retained secretions or packing rather than to inflammatory changes in the labyrinth. In these instances, when the fleeting symptoms have passed, there is little or no loss of hearing or of irritability of the static labyrinth. On the other hand, when the disturbances last, as they usually do, from several days to two weeks, there is frequently some loss of function when they have disappeared.

While the labyrinthine symptoms are at their height

there is never a total loss of function, i. e., there is either some hearing or the static labyrinth responds to at least one of the functional tests. If the caloric reaction is negative the hearing is, as a rule, destroyed. If there is no response to the caloric test, rotation may still arouse the diseased labyrinth, and when this too fails the fistula test is positive if a fistula is present.

By the fact that some function is retained, a diffuse serous labyrinthitis differentiates itself clearly from the suppurative stage. This differentiation, of course, is artificial, but clinical experience has shown that so long as a labyrinthitis is serous, i. e., so long as some function remains, extension of the infection to the intracranial contents, while it may occur, is exceedingly rare. Moreover, even after the inception of the purulent stage, i. e., when all function has been destroyed, there is a short time, varying between several hours and several days, before the labyrinthine infection extends to the meninges. In this interval, since the labyrinthine functions are destroyed, we are unable to estimate the progress of the disease.

When we consider that at present, the only means at our command to differentiate between the comparative safety of a serous labyrinthitis and the imminent danger of the suppurative stage are the functional tests, we readily appreciate the importance of carefully making these tests in every instance where the radical mastoid operation is contemplated or where, after the operation has been performed, labyrinthine symptoms supervene. A case illustrating the value of the functional tests is reported by *Neumann*. A radical mastoid operation was performed for the relief of a chronic suppu-

rative otitis media of many years' standing. The functional tests before the operation showed that considerable hearing remained and that the static labyrinth was normally irritable. Two days after the operation there occurred, with a slight rise of temperature, vertigo, vomiting and nystagmus directed to the sound side. Functional examination now showed deafness, with complete loss of irritability of the static labyrinth of the operated side. Upon these data the labyrinth operation was undertaken. The promontory was found necrosed, and was removed by pressure with the chisel alone, without resorting to the use of the mallet. Undoubtedly there had existed here a necrosis of the labyrinth wall, while the labyrinth itself remained intact until the trauma incident to the operation, caused a rapid extension of the middle ear suppuration. The comparison of the tests before operation with those made at the time of the labyrinthine symptoms proved this beyond a doubt, and served, moreover, as a basis for the surgical procedures.

In contradistinction to diffuse serous labyrinthitis, the suppurative variety may be either manifest or latent. The two forms are vastly different.

In the manifest form the symptoms are most violent. There is sudden deafness, associated with severe disturbances in equilibrium, persistent vomiting and a rotatory nystagmus directed to the sound side. The patient is unable to walk unassisted and lies in bed on the side of his sound ear. The functional tests show total loss of hearing and of irritability of the static labyrinth.

In the latent variety of **diffuse suppurative**

labyrinthitis there are frequently no symptoms at all. Not only are there no symptoms at the time of the examination, but there is often absolutely no history of vertiginous attacks. In these latent diffuse suppurative cases we may occasionally elicit some history of slight disturbances if we question the patients very carefully. It must be remembered, too, that patients do not grasp the relationship between the attacks of nausea, vomiting and vertigo, and the diseased ear. Such symptoms, if they have occurred, have been ascribed to disturbances in other (particularly the digestive) organs, and are therefore not associated, in the patient's mind, with the ear trouble.

The functional tests show a complete loss of hearing. The caloric and fistula tests are negative. If the disease has existed a long time, and if the labyrinthitis has been followed by the formation of new bone filling the intralabyrinthine spaces, the rotation test, according to *Ruttin,* behaves peculiarly. The duration of the after-nystagmus to the sound side equals the duration of that to the diseased side, but both are shorter than normal. *Ruttin* ascribes this to the fact that the sound labyrinth compensates for the complete loss of function in the diseased labyrinth.

The diffuse latent suppurative form of labyrinthitis is frequently as dangerous as it is insidious. Here, again, the functional tests are of inestimable value, for *Hinsberg* ascribes more than half the meningitides which follow either polyp extraction or the radical mastoid operation, to intracranial extension of latent suppurative labyrinthitis. If the functional tests indicate a diffuse latent suppurative labyrinthitis, polyp ex-

traction, or even the radical mastoid operation, must be looked upon as incomplete operative interference. As such they are exceedingly dangerous.

Necrosis and sequestration of the labyrinthine wall, as a rule, give us the clinical picture of a latent diffuse suppurative labyrinthitis. This is, of course, not invariably true as the case of *Neumann* (cited above) and the cases reported by *Ruttin* show. However, if a chronic suppurative otitis media has led to necrosis and sequestration of the labyrinthine capsule, there is often associated with the deafness and loss of irritability of the static labyrinth, a facial paralysis. The determination of the presence or absence of sequestration can sometimes be based upon the presence or absence of facial paralysis. This determination is of great importance, for the surgical indications may rest upon it.

Thus if latent suppurative labyrinthitis has existed a long time and compensation—as shown by the rotation test—has taken place, we may expect one of two conditions in the labyrinth. Either necrosis and sequestration or a new bone formation which has filled out the intralabyrinthine spaces. If the latter has occurred the labyrinth operation would, of course, be contraindicated. If, on the other hand, sequestration of the labyrinth wall has occurred, the labyrinth operation is indicated, at least the removal of the necrosed bone and the establishment of efficient drainage is necessary. Here a facial paralysis, which was not of too long duration, would act as a guide, since such a paralysis would exist only with necrosis and sequestration.

Even though the occurrence of disturbances of balance, loss of hearing, vomiting and nystagmus are

strongly suggestive of the most common cause of such symptoms, viz., labyrinthitis, it must not be forgotten that other conditions too may arouse almost identical phenomena.

It is possible for retained secretions in the middle ear to cause labyrinthine symptoms by increasing the intralabyrinthine pressure. If this occurs during an acute otitis media, while the membrana tympani is still intact or before efficient drainage has been established, a free paracentesis will bring speedy relief. The passing labyrinthine disturbances, which occasionally ensue after a radical mastoid, have also been ascribed to an increase in the intralabyrinthine pressure due to retained secretions or to gauze too firmly packed into the middle ear.

Labyrinthine inflammation must be differentiated from hysteria, affections of the eighth nerve, meningitis and abscess or tumor of the cerebellum.

From hysteria, a labyrinthitis may sometimes be differentiated by the condition of the middle ear, which in the former affection may show no pathological changes. If nystagmus is present in hysteria, it is not of the vestibular type, nor are the disturbances in equilibrium such as are caused by labyrinthine disease. In hysteria the body may have a tendency to fall backwards or forwards, or to either side, i. e., it follows no definite rule, while in labyrinthitis the tendency to fall is always in the direction of the slow component of the nystagmus, whatever the position of the head. In hysteria deafness may be simulated, but the static labyrinth will react to the functional tests.

The symptoms that occur in affections of the eighth

nerve simulate those of labyrinthine inflammations. Sometimes disease of the nerve develops in the presence of an apparently normal middle ear.

Under such circumstances, of course, an infective labyrinthitis, unless it be metastatic, can be excluded. Rapidly developing signs of stimulation or destruction of either the cochlear end-organ or the end-organs in the static labyrinth, alone and not in combination, indicate a retrolabyrinthine affection rather than endolabyrinthine disease. On the other hand, rapidly developing signs of stimulation or destruction of both the cochlear end-organ and the end-organs of the static labyrinth indicate endolabyrinthine disease rather than retrolabyrinthine affections.

In the differentiation between labyrinthine disease and affections of the eighth nerve, the bilateral simultaneous caloric and galvanic tests, as described by Ruttin, are of great value. By means of the caloric test we determine the comparative susceptibility of the two labyrinths, and, on the other hand, by the galvanic test we compare the irritability of the two vestibular nerves. Unfortunately the application of the galvanic current is frequently very painful, so that it cannot be used in every instance.

Those cases of polyneuritis in which both divisions of the eighth, as well as the facial nerve, are involved, are probably due to syphilis, and a positive Wassermann reaction may be present.

If a diseased labyrinth is exenterated, the nystagmus to the sound side, if any is present, will disappear in a short time. If a nystagmus directed to the side of the destroyed labyrinth develops after the labyrinth opera-

tion, it indicates either disease of the opposite labyrinth, meningitis or cerebellar abscess.

By the functional tests disease of the opposite labyrinth can readily be excluded. If this, then, is normal, and other signs of intracranial involvement are present, incision of the dura in the posterior cranial fossa, perhaps combined with exploration of the cerebellum, is indicated.

When the labyrinth has been destroyed by disease, but has not been operated upon, the diagnosis of labyrinthitis, to the exclusion of meningitis and cerebellar abscess, is not always so simple.

With a meningitis, however, the patient usually has a higher temperature than if the labyrinthitis is uncomplicated. With meningitis there is also restlessness and irritability, headache, rigidity of the neck muscles and tenderness upon pressure over the cervical vertebræ. Kernig's sign may be present, and there may be paralysis of the ocular muscles, and disturbances in the pulse rate and in respiration. The cerebro-spinal fluid is turbid, may be under pressure and may contain a large number of polynuclear leucocytes and bacteria. A meningitis may be diagnosed early in its course by means of the bio-chemical tests of the cerebro-spinal fluid, as described by *Kopetzky*.

With a cerebellar abscess there is persistent, severe headache, either frontal or occipital. There may be ataxia of the cerebellar type, with a hemi-paresis on the side of the lesion. The body may have a tendency to fall toward the diseased side. The direction in which the body falls is independent of the nystagmus, if this is present, and does not change when the posi-

tion of the head is changed. The temperature may be normal, subnormal or slightly elevated, but the pulse, as a rule, is abnormally slow. The patient appears to be severely ill. There may be nausea and changes in the eye grounds. In cerebellar abscess the balance disturbances do not diminish in severity, while in labyrinthitis the severity of the symptoms diminishes almost from their inception. The duration of the nystagmus, if this is present, may vary.

With a labyrinthitis the "pointing by" is in the direction of the slow component of the nystagmus. With a cerebellar abscess there is a loss of the normal reaction movements, particularly in the arm of the affected side. There is a destruction, centrally, of the tonus of those muscles which tend to draw the arm of the affected side inward, or toward the other side. Thus if an abscess exists in the left lobe of the cerebellum, the left arm will "point by" to the left spontaneously, and independent of the nystagmus, if the latter is present.

If the pointing tests are made, after syringing the right ear with cold water (causing nystagmus to the left), the left arm will not deviate in the direction of the slow component, i. e., to the right, because the cerebellar centre or centres that influence the movement of the left arm inwards or to the opposite side have been destroyed by the abscess. This loss of normal reaction movements may not be confined to the shoulder joint, but may extend to the elbow, wrist and lower extremity as well.

As we have stated before, the vestibular nucleus of either side is in relation with the central nuclei of the muscles of both eyes. When an impulse from either labyrinth reaches its nucleus, there arise in the latter,

secondary impulses which extend to the nuclei of the eye muscles. Those secondary impulses which result in nystagmic movements of the eyes, directed toward the side of the labyrinth in which the primary impulse was aroused, are called homolateral, and those impulses through whose influence a nystagmus in the opposite direction is aroused, are called heterolateral.

Through the medium of the cerebellum, the influences of all the other sources of balance impulses, i. e., the checks and balances, exercise a restraint upon the vestibular impulses. Each cerebellar hemisphere exercises this inhibitory influence upon those impulses which emanate from the vestibular nucleus of its own side.

Let us assume, for instance, that the left labyrinth has not been destroyed by disease, but that either a tumor or an abscess has formed in the left lobe of the cerebellum, in such a position that the restraining influences over the impulses from the left vestibular nucleus are destroyed. If, then, a primary impulse is aroused in the left labyrinth which produces chiefly heterolateral secondary impulses (i. e., through cold water irrigation), there will result a nystagmus directed to the right, which will be uninfluenced by the restraint exercised normally through the medium of the cerebellum. Such a nystagmus would last, therefore, much longer than normal. To this phenomenon Neumann has applied the term "enduring nystagmus," and he states that it lasts from 2½ to 15 minutes or even longer.

CHAPTER VI

TREATMENT OF LABYRINTHITIS

A large number of cases of labyrinthine disease recover without treatment. Many get well as the result of treatment, either operative or non-operative. A small proportion die from intracranial complications. Of the cases which recover, some have complete restoration of function, while in others there is partial or total loss of the functions of the internal ear. Of the fatal cases, the vast majority are of the diffuse purulent type. A few cases of fatal meningitis, complicating circumscribed purulent and diffuse serous labyrinthitis, have been reported.

Even cases of diffuse purulent labyrinthitis may heal spontaneously. This is true particularly of those labyrinthitides which complicate scarlet fever. Many histological examinations of the temporal bones of deaf-mutes have shown that a purulent process in the labyrinth may heal, and the intralabyrinthine spaces become filled by new bone. Far more frequent, however, than this spontaneous healing is the transition from the acute to the chronic stage, which may last for years. While in some instances no further trouble ensues, yet in the majority of these cases, sooner or later, serious complications develop. We must therefore consider every labyrinthitis, even in the latent stage, an extremely serious condition.

We may lay down as a broad, general rule, that in no case of circumscribed labyrinthitis should the inter-

nal ear be operated upon. The circumscription of the disease means that nature has been successful in erecting a barrier against the spread of the infection. If we operate in such a case, we may break down the barrier which nature has erected, and thus cause the process to extend into the cranial cavity. This is especially true of incomplete operations on the labyrinth. In complete labyrinth operations the breaking down of protective adhesions and granulations does not possess the same significance, because we provide for adequate drainage here.

Perhaps the most frequent cause of the spread of a labyrinthine infection to the intracranial contents is incomplete operative interference. The fresher the labyrinthine infection, i. e., the less time nature has had to wall off the disease, the more certain is the spread of the infection. In cases of manifest diffuse suppurative labyrinthitis the radical mastoid operation should never be performed alone. It is far better to leave these cases entirely alone than to perform the radical mastoid operation without, at the same time, establishing adequate drainage for the infected endolabyrinthine spaces.

Another reason for not opening the labyrinth, in circumscribed labyrinthine disease, is that by so doing we destroy the remaining labyrinthine functions.

In our attitude toward diffuse serous labyrinthitis we must also be influenced by the effect of our treatment on the functions of the labyrinth, for in this type of inflammation there is usually restoration of function after the disease has subsided.

It is of the utmost importance to positively establish

our diagnosis before operating on a case of labyrinthine disease. For to place a normal labyrinth into open communication with an infected middle-ear cavity invites certain infection of the labyrinthine contents. In a diseased labyrinth, on the other hand, inflammatory adhesions have, in most cases, shut off the infected labyrinth cavity from the intracranial lymph spaces before operation.

Fortunately we are now enabled, as the result of functional examination, to obtain a fairly accurate notion of the presence and the extent of inflammatory involvement of the internal ear.

Treatment of labyrinthitis may be of three kinds:

1. Non-operative treatment.

2. Operative treatment of the middle-ear spaces alone.

3. Operative treatment of the middle-ear spaces combined with the labyrinth operation.

The radical mastoid operation must, of course, precede the operation on the labyrinth. The two operations, however, may be performed at one sitting, or there may be a considerable interval between them.

In the treatment of certain forms of labyrinthitis there is a unanimity of opinion. In other forms, however, particularly in the acute (manifest) diffuse purulent form, there is still considerable difference of opinion as to the proper method of treatment.

The questions to decide are whether to operate and how to operate. It must be decided whether to do a radical mastoid operation or to do a labyrinth operation also; whether to do the two operations at one sitting or with an interval between them; whether to open

a portion or all of the labyrinthine spaces; whether merely to open the labyrinthine spaces or exenterate the entire labyrinth, with the portion of the petrous pyramid in which it is embedded. Finally, we must decide whether or not to explore the cranial cavity.

In cases of empyema of the labyrinth, where there is no bone involvement, opening the labyrinthine spaces is sufficient. When there is necrosis of a portion of the bony capsule a labyrinthectomy should be done. That portion of the petrous pyramid containing the labyrinth, from the sigmoid sinus behind, to the carotid canal in front, should be removed. In performing this operation the canal containing the facial nerve must be left intact if possible. When there are symptoms of intracranial involvement, it may be necessary to explore both the posterior and middle fossæ of the skull.

Alexander believes that no extensive operation should be done on the labyrinth without exposing the dura. He says that "an extensive opening of the labyrinth, without simultaneous exposure of the dura, is a very dangerous procedure, which exposes the patient to the possibility of a postoperative purulent meningitis." He states further that we should always explore the dura in the region of the saccus endolymphaticus, i. e., the posterior pyramidal wall between the sinus and the internal auditory meatus, because extradural abscess, without symptoms, is not at all uncommon in this region.

If during the labyrinth operation there is evidence that the disease has spread in the direction of the middle fossa, the dura of the middle fossa should also be exposed. If lumbar puncture shows a pathologically

changed cerebro-spinal fluid, and other signs of meningitis are present, the labyrinth operation must be combined with incision of the dura.

For the sake of convenience we will divide labyrinthitis into its clinical forms, and state the indications for treatment in each form. We will consider circumscribed labyrinthitis, diffuse serous labyrinthitis, diffuse purulent manifest labyrinthitis and diffuse purulent latent labyrinthitis.

Circumscribed labyrinthitis is usually determined by the ability to elicit a positive fistula test in the presence of more or less of the labyrinthine function. Hearing may or may not be present. The caloric test is usually positive, and the rotation test normal. Complete deafness does not necessarily indicate a diffuse labyrinthitis, for the destructive changes in the end-organ or nerve may be the result of toxic or degenerative atrophic processes.

If the circumscribed labyrinthitis occurs during the course of chronic suppurative middle-ear disease, as is most commonly the case, a radical mastoid operation should be done. There is no urgency in doing the operation, as the labyrinthine disease may remain circumscribed indefinitely. But as these middle-ear suppurative conditions are usually complicated by cholesteatoma, it is useless to treat them non-operatively.

On exposure of the middle-ear spaces, during the radical mastoid operation, we usually see the fistula, whose most common location is on the eminence of the external semicircular canal. The fistula should neither be probed nor curetted.

The patient should be watched very carefully after

the operation for the possible development of a secondary diffuse serous or purulent labyrinthitis. The trauma of the radical mastoid operation is sometimes responsible for the transformation of a circumscribed into a diffuse labyrinthitis. This extension usually manifests itself on the second or third day following the radical mastoid operation, and is recognized by loss of hearing, vertigo, nausea and vomiting with a combined rotatory and horizontal nystagmus in the direction of the sound ear. If a spontaneous nystagmus was present before the operation, it was probably directed toward the side of the diseased ear, or toward both sides in accordance with the position of the eyes.

In order to determine whether the diffuse labyrinthitis is of the serous or the purulent type, a functional examination must be made. In the serous form there is usually some remnant of function left.

If the patient hears loud speech through the bandage, the sound ear being excluded by the noise apparatus, we can make a positive diagnosis of serous labyrinthitis. If he does not hear, even with the bandage removed, we do a caloric test, using hot (112° F.) sterile salt solution. If this does not neutralize or reverse the spontaneous nystagmus, and if the fistula test (which is made by placing a cup over the wound and the auricle) is negative, we are dealing, in all probability, with a purulent labyrinthitis. Rarely we have complete destruction of function with diffuse serous labyrinthitis, but such cases are treated as if they were purulent.

These cases, particularly if there is any rise of temperature, should be operated on immediately, the laby-

rinth being opened wide, in order to prevent the spread of the infection into the cranial cavity.

In cases of diffuse serous labyrinthitis, we do not operate. These cases usually subside in a week or two. Cases of induced serous labyrinthitis are also treated without operation, but they must be carefully observed, and if complete loss of function supervene during the active stage, the labyrinth must be opened.

Sometimes a labyrinthine fistula refuses to heal after the radical mastoid has been performed. The radical cavity epidermatizes in its entire extent except for a small spot at the fundus, which continues to granulate. Such a condition is usually due to the presence of a necrotic area in the bony capsule about the fistula. A secondary radical may be performed and the necrotic bone removed. A complete labyrinth operation, of course, should not be done in such a case.

If the circumscribed labyrinthitis occurs during the course of an acute middle-ear suppuration (which is very uncommon) a simple mastoid operation is usually sufficient to cure the condition.

The cases of diffuse manifest purulent labyrinthitis are recognized by the occurrence of a spontaneous rotatory and horizontal nystagmus toward the sound ear, vertigo, nausea and vomiting and a complete absence of labyrinthine function. The patient is absolutely deaf, and there is a negative caloric and fistula test.

There is still some difference of opinion in regard to the treatment of this form of labyrinthitis. *Neumann* and *Ruttin* believe that these cases must be operated on at once, the labyrinth operation being done in one sitting with the radical mastoid. *Alexander,* on the other

hand, holds that they should be operated on only if they show signs of beginning intracranial complications, such as sleeplessness, headache, fever and rigidity of the neck. He believes that these symptoms appear as soon as the suppuration in the labyrinth has become diffuse. In the cases without signs of endocranial involvement, he advises complete rest in bed, and conservative treatment of the middle-ear suppuration.

Ruttin, on the other hand, believes that every case of manifest purulent diffuse labyrinthitis, with complete loss of function, should be operated on immediately; for in the absence of all labyrinthine function we have no indicator to warn us of the progress of the infection.

Of 20 such cases of diffuse purulent manifest labyrinthitis, operated on at *Urbantschitsch's* clinic, and reported by *Ruttin,* 12 recovered and 8 died. Of the 8 fatal cases 5 already had signs of meningitis at the time of operation, i. e., they were either comatose or had purulent cerebro-spinal fluid. This leaves 3 out of 20, or 15% in which the operation failed to prevent the spread of the infection to the meninges. However, there are cases which recover, despite the fact that meningeal symptoms are already present at the time of operation.

In the cases of latent diffuse purulent labyrinthitis, there are no symptoms, except those of the chronic middle-ear suppuration. But on examining such a case, it is found that all of the labyrinthine functions have been lost. There is no hearing. Caloric and fistula tests are negative.

These cases are very treacherous. There may be no

symptoms for years. But at any time, as the result of an acute exacerbation of the middle-ear suppuration, or as the result of an operation on the middle ear or mastoid, there is a sudden extension of the inflammation to the meninges.

In these latent cases there is usually no haste necessary, but, if operated at all, a complete labyrinth operation must be done. A radical mastoid operation in such a case is very dangerous. It is very apt to cause a lighting up of the labyrinthine infection, with extension to the meninges.

In every case of chronic suppuration of the middle ear it is absolutely imperative to test the labyrinthine functions before doing a radical mastoid. If it is found that the labyrinthine functions have been destroyed, either a labyrinth operation must be done or no operation at all. Of 25 cases of latent diffuse purulent labyrinthitis reported by *Ruttin,* 21 recovered and 4 died.

Every case of chronic middle-ear suppuration with loss of labyrinthine functions, is not necessarily a latent labyrinthitis. We may be dealing with a healed labyrinthitis. Unfortunately we cannot be absolutely sure whether the labyrinthine inflammation is latent or healed. But there are certain points which help us considerably in our decision.

It has been found by *Ruttin* that after complete destruction of the labyrinthine contents of one ear, followed by fibrous or bony healing, the rotation test behaves peculiarly. The after-nystagmus toward the side of the diseased ear is as long as that toward the opposite side, but both are shorter than normal. This he calls the compensation symptom.

If in a chronic suppurative middle-ear condition, the functional examination shows complete destruction of the labyrinth, and the presence of the compensation symptom, and there are evidences of new bony growth on the inner tympanic wall, especially in the region of the external semicircular canal, then we are, in all probability, dealing with a healed labyrinth, and not a latent labyrinthitis. In such a case, it would naturally be unnecessary to open the labyrinth.

In cases of suspected latent labyrinthitis, *Neumann* is guided by the character of the bone in the petrous pyramid, and the conditions found in the posterior cranial fossa. If the petrous pyramid shows marked pneumatization of the bone, or if there is a deep extradural abscess in the posterior cranial fossa, he concludes that there is a suppuration in the labyrinth.

Politzer is guided largely by the behavior of the wound after a radical mastoid. If there are persistent exuberant granulations on the inner tympanic wall, after the radical mastoid operation, he assumes the presence of a labyrinthitis.

The occurrence of a facial paralysis in the course of a chronic middle-ear suppuration, when the functional tests show a non-functionating labyrinth, is positive evidence of a latent labyrinthitis.

A typical labyrinth operation for all cases of labyrinthitis is impracticable. We may divide the operations on the labyrinth into two groups. In one group, the operation is limited to an opening of the labyrinthine spaces, or labyrinthotomy. In the second group, the operation consists of the removal of the entire labyrinth, as far as practicable, with the adjacent portions

of the petrosa. This latter type of operation may be called labyrinthectomy. In the former class belong *Richards', Hinsberg's* and *Bourguet's* operations, and in the latter class, *Jansen's* and *Neumann's*. In the first type of operation, the great danger is injury to the facial nerve, and in the second class, injury to the dura.

A simple enlargement of a labyrinthine fistula is of no value for draining an infected labyrinth. A wide opening of the labyrinthine spaces behind and in front of the facial canal is indicated in empyema of the labyrinth, with intact bony capsule. When there is involvement of the bony capsule, we must do a labyrinthectomy, with removal of that portion of the petrous pyramid which contains the labyrinth. When there are signs of intracranial involvement, the posterior fossa of the skull must be explored, and sometimes the middle fossa.

The question as to whether the cochlea should be opened together with the vestibule, in every case in which the labyrinth is operated on, is still the subject of discussion. But since we open the labyrinth only in cases of diffuse labyrinthitis, it seems but rational that the cochlea should be opened as well as the vestibule, in every case. It is usually unnecessary to do more than to uncover the lower half of the basal whorl of the cochlea. This can be done with one stroke of the chisel, and if a reasonable amount of care is exercised, is a perfectly safe procedure. In addition to draining the cochlea, this procedure helps to drain the vestibule.

In sequestration of a large portion of the labyrinth, the sequestrum is removed, and the remaining portions

of the labyrinth left intact, in order not to destroy the protective granulation-zone set up by nature.

In regard to the treatment of injuries to the labyrinth during the performance of a radical mastoid operation, there is some difference of opinion. *Alexander* believes that if the labyrinthine cavity is opened accidentally during the radical operation, the entire labyrinth should be widely opened, for the same reason that a small accidental tear in the dura should be widely opened. He believes that such injuries to the labyrinth, if left untreated, almost always end fatally. *Jansen,* on the other hand, believes that these cases should be left alone. He has found that most of the cases get well.

The most rational course to pursue, it seems to us, is to treat the case expectantly, until signs of a diffuse purulent labyrinthitis have supervened, and then to operate on the labyrinth. Of the cases of operative injury to the labyrinth seen by the writers, the cases of injury to the semicircular canals have gotten well without operation on the labyrinth. Of three cases of dislocation of the stapes, one recovered, without operation, and two died in spite of operation on the labyrinth.

In tuberculous labyrinthine disease, operation is indicated only when the formation of a sequestrum demands it.

In meningogenic labyrinthitis, opening the labyrinth is only of value to preserve hearing. Whether it ever does this is problematical.

Non-operative treatment of labyrinthitis consists principally of rest in bed, to minimize the unpleasant subjective symptoms. The patient usually finds the

PLATE XX.

Fig. 71

Hinsberg's Operation on the Labyrinth

Oval window is enlarged downward
Vestibule is entered behind facial ridge, through anterior limb of external semicircular canal

most comfortable position for himself. It is the position in which nystagmus and vertigo are least marked, and is usually on the side of the sound ear. The patient should be left in bed as long as there is any vertigo. This may be anywhere from three or four days to several weeks. Rest in bed may be of some value to prevent the spread of a labyrinthine infection.

Galvanization has been employed to relieve the vertigo. One electrode is placed in front of each ear. If the spontaneous nystagmus is to the right, the kathode is placed in front of the left ear, and the anode before the right ear. A current of 4 to 12 milliamperes is usually sufficient to stop the nystagmus and the accompanying vertigo.

Bromides and other sedatives may have some value.

During this time there should be no manipulative interference with the middle ear. It may be irrigated with warm antiseptic solutions.

HINSBERG'S OPERATION

Of the various operations on the labyrinth which have been described up to the present, the most conservative is that of *Hinsberg*. His operation consists of the removal of the outer wall of the vestibule, in front of and behind the facial canal, and in this way establishing drainage. The semicircular canals are not touched, except that portion of the external semicircular canal which must be removed in order to reach the vestibule. The cochlea is drained by removing the outer wall of the lower half of its basal whorl (Fig. 71, Plate XX).

The steps of the operation are as follows:

First a radical mastoid operation is done, the posterior canal wall being shaved down as far as possible without jeopardizing the integrity of the facial nerve. The posterior canal wall is removed as far as is necessary to make the oval window easily accessible.

If the stapes is present, it is removed. The oval window is now enlarged at the expense of its lower margin. This may be done either by means of a chisel placed across the bridge of bone between the oval and round windows, or with a burr. When a chisel is used it is directed downward, and removes the bridge of bone between the oval and round windows. When a burr is used, it is placed against the lower margin of the oval window, care being taken not to injure the inner vestibular wall. The posterior margin of the oval window must not be touched, in order to avoid injuring the facial nerve. The oval window may be enlarged to twice its normal size.

Sometimes *Hinsberg* removes the promontory. This exposes the lower half of the basal whorl of the cochlea.

The external semicircular canal is then opened. *Hinsberg* prefers to use a drill for this work. The ampullated end of the external semicircular canal is nearer to the antrum than is the small end. The point where the outer wall of the external semicircular canal is thinnest, according to *Hinsberg,* and where it is easiest to enter, is in the region vertically above the fenestra ovalis. From here, the anterior limb of the external canal is followed in an inward and forward direction, until the vestibule is reached. In order to determine when the vestibule has been opened, a bent probe is

passed through the enlarged oval window. Its end will appear in the cavity behind the facial ridge.

In cases where there is a deep overhanging middle-fossa, it is difficult to open the external semicircular canal at its anterior part. In these cases, *Hinsberg* advises opening the vestibule from behind, after the manner of *Jansen.*

RICHARDS' OPERATION

Richards first opens, and then removes all three semicircular canals, and opens the vestibule behind the facial nerve, through the solid angle. He then opens up the entire cochleal cavity, and removes the apex of the modiolus. The operation is best described in his own words.

"A *Schwartze-Stacke* operation is done, by which we secure the maximum amount of working room, and in which we exenterate the bony angle included between the groove of the sinus knee and the under surface of the middle fossa, which better enables us to work in the axis of the petrous pyramid. We lower the facial ridge to its absolute limit, remove the fringe of bone on the anterior aspect of the facial ridge back to the descending limb of the facial nerve, remove the outer wall of the hypotympanum, and lower the level of the canal floor, securing by these steps the maximum exposure of the outer wall of the vestibule, and the dome of the jugular bulb should it rise high.

"As exploration of the cochlea may be necessary, it is important that we see the exact position of the carotid artery by the exposure of its canal. In consequence

we shave down the convexity of the anterior wall of the auditory canal, remove the lip of bone overhanging the mouth of the tube and evulse the tensor tympani.

"This exposes the tube to curettement, by which we relieve the field of blood, and it gives the maximum width to the apex of the cavity. Should the consistency of the bone permit, the arches of the semicircular canals should be delineated. This enables us to work with accuracy. The cavity should be cleansed; the tube packed with adrenalin gauze; the field rendered bloodless; the instruments and hands resterilized.

"We next remove the prominence of the horizontal semicircular canal; it is a treacherous structure (Fig. 72, Plate XXI). The cutting edge of the chisel is placed at a point below the summit, but well above the level of the fallopian canal, for this prominence separates along definite planes of cleavage, and as the outer lip of this semicircular canal is intimate with the fallopian, a fissure in the former may extend out into the latter. Should the plane of cleavage be on a level with or below the fallopian canal, the facial nerve may either be exposed by having its roof removed, or else completely undermined, under which circumstances we may definitely expect paralysis, for it will ensue. The stroke should be made in a direction corresponding to the plane of the canal. The remaining canals are next uncapped, and the condition of the interior is noted. It will be found that the interior of the external semicircular canal most frequently of all shows pathological change. In opening the superior canal a curved gouge should be used. It permits the stroke to be made in the

PLATE XXI.

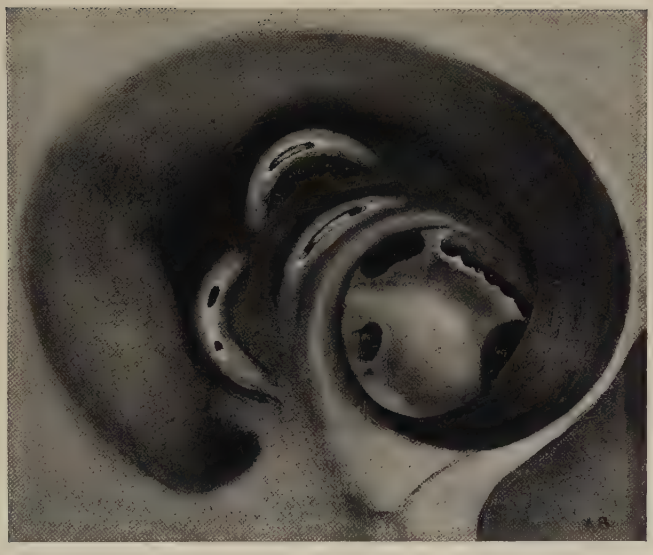

FIG. 72

RICHARDS' OPERATION ON THE LABYRINTH

Radical mastoid operation has been done. The three semicircular canals have been outlined, and uncapped, opening up their lumina

PLATE XXII.

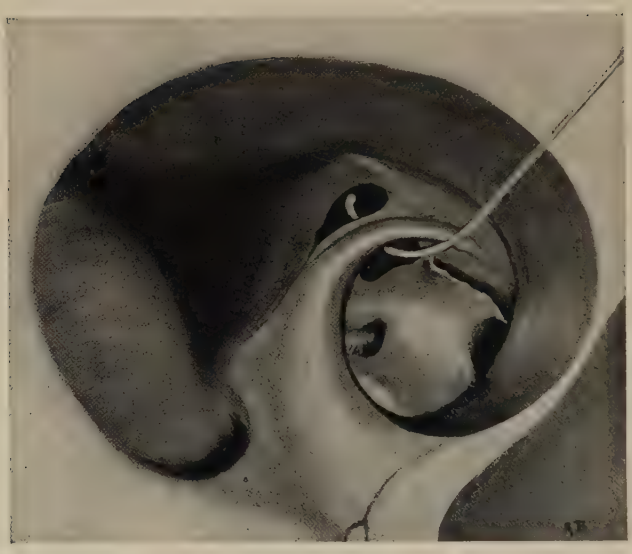

Fig. 73

Richards' Operation on the Labyrinth

Semicircular canals have been removed, and vestibule opened from behind, leaving the ridge containing the facial nerve intact. Probe has entered the vestibule through the oval window

direction of safety. The danger arises from the non-support of this canal by solid bony structure.

"Owing to the contrast between the dark interior of the canals and the brilliantly illuminated white bone surrounding them, the labyrinthine fluid may appear dark and be mistaken for blood or granulations; we should not be deceived by this illusion.

"We next enter the vestibule through the solid angle of the semicircular canals by creating at this point a conical pit with its apex directed inward and gradually lowered until it enters the vestibule (Fig. 73, Plate XXII). As it is necessary to remove the inner lip of the horizontal semicircular canal, during this step the chisel should be held perpendicular to the plane of cleavage, and under no circumstances should it impinge upon the outer lip, which is intimately associated with the fallopian canal, together with which it is left as a bridge carrying the nerve which spans the vestibule.

"The opening in the vestibule is now enlarged until a full exposure is obtained of this portion of the cavity. Its inner wall should be searched for fistulæ. In enlarging this cavity no pressure should be made upon the bridge. For this reason a curette is a dangerous instrument. A small sharp gouge is safer.

"Owing to widespread necrosis, it is necessary in some instances to sacrifice the bridge of bone carrying the facial nerve. To accomplish this without injury to the facial, we select a curette with its cutting edge turned backward, utilize the superior rim of the bone cavity as a fulcrum and shave off from above downward in a direction parallel to the course of the nerve, the roof of the fallopian canal, thus leaving the nerve ex-

posed in its gutter of bone, from which it may be separated and lifted without injury. Any filaments given off from the nerve should be cut and not torn from this structure, as unnecessary traumatism is committed. We next remove such portions of the bridge as are necrotic, but no more, for the nerve here represents a curve, and should it lose the entire support of its bony gutter, it apparently elongates, and consequently sags or kinks, and, becoming enmeshed in this vicious position by the granulations, has its functions interfered with later.

"In this condition the nerve stretches as an exposed structure from its point of emergence low down on the facial ridge, across the cavity of the vestibule to its entry into the inner wall of the tympanum, corresponding to a point above and anterior to the original site of the oval window.

"In knocking out the inner edge of the bony ridge, which corresponds to the upper inner wall of the tympanum, the stroke of the chisel should not be made from behind forward in a direction parallel to the course of the nerve, but from above downward, or, from before backward; i. e., in a direction perpendicular to the course of the nerve and to the fallopian canal.

"The reason for this is that the bone at this point tends to separate when struck from behind forward, along a plane of cleavage which crosses the fallopian canal, causing its fracture; as the fallopian canal at this point represents the greater portion of an exposed cylinder, its fracture results in the facial nerve being encompassed by a little annulus or cylinder of bone, which may be slid up and down upon the nerve, just as a

ring upon a finger. This complication is—so far as the integrity of the nerve is concerned—a formidable one, and it requires the greatest patience to remove the little annulus without causing injury to the facial. To accomplish it, we steady the annulus with mouse-tooth thumb forceps and with a pair of rongeurs—the jaws of which are accurately apposed—crush it in a direction parallel to the course of the nerve.

"To avoid this complication, it is only necessary before attempting the removal of the upper portion of the bony ridge that the fallopian canal should have been thoroughly converted into a gutter, by shaving off its roof, as mentioned, and by making the stroke in the direction indicated. Should the accident now occur, the nerve can be easily freed from the little mass of bone by slipping it through the open side of the annulus, or cylinder.

"Should we have to expose the nerve as mentioned, no attempt should be made to retract it or draw it to one side for the sake of gaining room for manipulation, as this is unnecessary; for the fallopian canal at the points at which the nerve emerges from and enters the bone is represented by sharp, serrated margins, against which if the nerve is drawn it may be lacerated at its fixed points. The sharp spicules should be removed so that the margin of the canal at these respective points is represented by an even rim.

"The next step is to expose the anterio-inferior cavity of the vestibule by removing the posterior aspect of the promontory and the outer vestibular wall. We select a gouge, the width of which corresponds to the distance between the round and oval windows. The cut-

ting edge straddles the little bridge of bone separating these two openings, and the stroke—which is slight, for the posterior aspect of the promontory is brittle and fractures easily—is directed from above, downward and forward in the direction of the first cochleal turn. We must at this step think of the possible position of the dome of the jugular bulb.

"The greatest care should be taken that the cutting edge of the chisel does not cross the cavity of the vestibule and impinge upon its inner wall, for the inner wall at this point bulges outward and is separated from the internal auditory meatus by a brittle partition of bone not more than $\frac{1}{32}$ of an inch in thickness. Should the inner wall be fractured, we lose cerebro-spinal fluid and aside from the inconvenience caused by this accident, the infected operative cavity is placed in direct communication with the intracranial cavity and the patient's life is jeopardized; meningitis will probably result.

"As the cerebro-spinal fluid is under pressure and is of low specific gravity, gauze does not act as an efficient plug to the rent; sterile wax is preferable.

"By enlarging this opening we secure, with the previous steps, the full exposure of the vestibular cavity and the beginning of the first cochleal whorl. Should we now find that the disease has invaded the cochlea, we must continue the exploration of the cochlea until we have followed the diseased process to its legitimate end.

"We next remove the roof of the first cochleal whorl from behind forward, exposing its interior to a point just short of the carotid eminence (Fig. 74, Plate

PLATE XXIII.

FIG. 74

RICHARDS' OPERATION ON THE LABYRINTH

A portion of the promontory has been removed, exposing the beginning of the basal whorl of the cochlea

FIG. 75

RICHARDS' OPERATION ON THE LABYRINTH

Exposure of lower half of basal whorl and second whorl of the cochlea

XXIII). The instrument of preference is a thin, sharp gouge with no shoulder, the width of which is slightly greater than that of the cochleal whorl. Four structures are to be avoided; the dome of the jugular bulb below, the eminence of the carotid canal in front, the base of the modiolus and the internal auditory meatus internally. The danger to the first two is slight; to the second two it is imminent, and injury to these structures is to be avoided only with the exercise of care.

"By confining the removal of the cochleal shell entirely to the roof of its first whorl and not allowing the gouge to impinge upon the inner wall of the cochleal turn these last dangers may be averted. From the carotid artery in front, the cavity of the first cochleal turn is separated by a hard cuff of bone which serves the purpose of an efficient bumper; though thin this partition is sufficient.

"If we now find that the limit of the disease has not been reached, we must explore the remaining cochlea. This constitutes by far the most difficult and dangerous step of the procedure; for the cochlea, which represents an extremely small cavity encased in a brittle shell of bone, is hemmed in on all sides by structures which we cannot afford to injure. In front is the carotid artery, below the dome of the jugular bulb, behind the internal auditory meatus, above and behind in immediate proximity to the second half of the first cochleal turn, the knee of the facial nerve. A circle a quarter of an inch in diameter could be so placed as to pass through the majority of these structures.

"Were the above factors the only ones to be considered it would be comparatively easy to select a point

on the cochleal shell which from dead house work we had found to be a safe one, and open its cavity. But the difficulty lies in the fact that within this shell of bone is contained a structure which from its position is exposed to injury, and which from a surgical standpoint is the most treacherous of the internal ear; I refer to the modiolus.

"Before approaching the cochlea it will be well to consider certain anatomical features of the modiolus which are of surgical importance. The modiolus represents a small pyramid of bone seated upon the internal auditory meatus, decreasing in size from base to apex. Its apex is its weakest point, but the next weakest point is not immediately below its apex, but at the extreme base; for its base is excavated by the internal auditory meatus, and the pyramid consequently rests upon a mere rim of thin, brittle bone. If the chisel is applied to the pyramid well above its base and a stroke made, the fracture does not take place at the point of applied violence but at its base; when this occurs the pyramid fractures completely round the circumference of its base and separates as a single piece of bone. The internal auditory meatus is consequently opened throughout its entire circumference, and as the diameter of the base of the pyramid or the rim of bone upon which it rests is about ⅛ of an inch, the loss of cerebrospinal fluid is rapid. The failure to appreciate the surgical importance of this anatomical feature of the modiolus resulted in the death of the first patient.

"If we now examine the modiolus with a strong convex lens we see that the pyramid has an outer casing of brittle bone and a core which is porous, made so by

canals running from base toward apex for the passage of various structures. These canals are not completely filled by the structures which they contain, and this permits the cerebro-spinal fluid to penetrate out into the modiolus.

"During the second operation in which the modiolus had been removed well down toward its base it was noticed that a sepage of cerebro-spinal fluid took place through its stump; the intracranial cavity had been placed in gross communication with the infected cavity in an altogether unexpected way through the aforementioned channels, and the failure to appreciate the significance of this structural character of the modiolus resulted in the death of the second patient. In this case the infection could be traced along the cochlear branch of the auditory nerve. It now became important to determine how far down from the apex toward the base the pyramid could be removed without putting the intracranial and operative cavities in gross communication; for in dealing with the anterior half of the cochleal cavity it is absolutely necessary to get rid of a portion of the modiolus.

"It can be shown upon increasing the tension of the intracranial fluid in a cadaver by injection, or in the living subject by pressure over the internal jugular vein, under which circumstances the cerebro-spinal fluid will penetrate as far out as possible into the modiolus (i. e., grossly), that the pyramid may be removed from the apex toward its base, down to a point corresponding to the termination of the first cochleal whorl without causing the loss of cerebro-spinal fluid; i. e., without placing the operative and intracranial cavities

in gross communication. As will be shown later, this suffices for the complete exploration of the anterior half of the cochleal cavity.

"In approaching, therefore, the cochlea, we must remove its shell in such a way as not to injure the modiolus. We select a point in the cochleal shell corresponding somewhat to the apex of the cochleal cavity, and with a thin sharp gouge shave it down until the dark interior of a cochleal whorl shows through the thin lamella of bone (Fig. 75, Plate XXIII). The stroke should be made from above downward and forward in a direction corresponding to that of a cochleal whorl.

"Not infrequently the shell of the cochlea is scalloped and the position of the cochleal turns roughly indicated. In this way a window is created in the cochleal shell, and we now enlarge this window, completely exposing the upper portion of the cavity.

"In enlarging this window the small gouge is the instrument of preference; an attempt to insinuate a very fine curette beneath the opening in the cochleal shell causes its back to impinge or press upon the modiolus, which sticks up as a little tent pole in the cavity, and this may result in the fracture of the pyramid at its base. The gouge merely removes the shell without endangering the pyramid.

"To expose the second half of the first cochleal whorl it is necessary to remove the apex of the pyramid down to a point corresponding to the termination of the first cochleal whorl (Fig. 76, Plate XXIV). This done, we can look down over the stump of the pyramid, upon the roof the second half of the first whorl, and with a small gouge carefully break through its roof from above, ex-

PLATE XXIV.

FIG. 76

RICHARDS' OPERATION ON THE LABYRINTH

Lower half of basal whorl, middle whorl, and cupola of cochlea exposed

PLATE XXV.

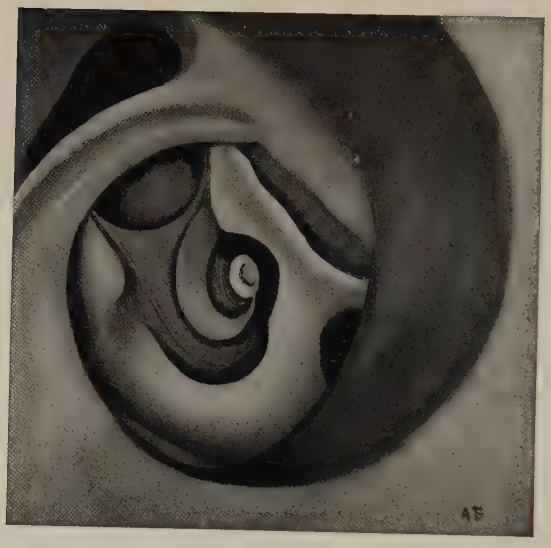

Fig. 77

Richards' Operation on the Labyrinth
Exposure of the entire cochleal cavity

PLATE XXVI.

Fig. 78

Richards' Operation on the Labyrinth

Complete exenteration of the cochlea. The stump of the auditory nerve is seen at the bottom of the cochleal cavity

posing in this way the entire interior of the cavity of the cochlea (Fig. 77, Plate XXV). In breaking through the roof over the last portion of the first whorl, we are in direct relation to the facial nerve and the internal auditory meatus, both of which must be avoided." (Fig. 78, Plate XXVI).

THE JANSEN OPERATION

Jansen operates within the temporal bone, except where a diagnosis of disease in the posterior cranial fossa has been made. In the latter case, he exposes the dura of the posterior fossa, and removes the posterior surface of the petrous pyramid. The former method is called the semicircular canal or tympanal method, and the latter the endocranial method.

The *Neumann* operation is just like *Jansen's* endocranial operation, except that *Neumann* continues the removal of the posterior pyramidal surface until the posterior wall of the internal auditory canal has been removed, and the auditory and facial nerves lie exposed in the canal. *Neumann* believes that the internal auditory canal should be opened in every operation on the labyrinth, on account of the frequency with which an abscess is found at the fundus of this canal.

Jansen's semicircular canal or tympanal method is as follows; with a chisel or burr he removes the eminence of the external semicircular canal. The upper wall of the anterior limb of the external semicircular canal is then removed. Great care must be exercised not to injure the lower wall of this canal, as immediately below it lies the facial nerve. He then removes

the entire posterior limb of the external semicircular canal, together with the underlying bone, in a downward and backward direction, being careful not to injure the facial nerve in front. By following the anterior and posterior limbs of the external semicircular canal, the vestibule is reached. A bent probe passed into the vestibule through the oval window is a valuable guide during the operation.

After reaching the vestibule, he enlarges the opening into it until the entire outer vestibular wall, behind the facial nerve, is gone. He removes the outer wall, in a downward direction, until the ampullary orifice of the posterior semicircular canal is seen; backward, until the orifice of the common limb is seen; and upward, until the ampullary orifice of the superior semicircular canal is exposed.

He does not follow up the semicircular canals. He believes that the disease in the canals is usually slight, and that after exposure of their orifices into the vestibule they are very likely to heal.

In cases of narrowing of the vestibule and obliteration of the semicircular canals, resulting from the formation of new bone, he penetrates the vestibule behind and parallel to the facial ridge, inward downward and forward, in the direction of the fenestra ovalis.

Jansen believes that while the chisel gives one a better idea of the contents of the labyrinthine spaces, as the canals do not fill up with bone-dust, the burr, on the other hand, is less dangerous to the integrity of the facial nerve. He uses the chisel as a plane, thus taking off successive shavings of bone.

PLATE XXVII.

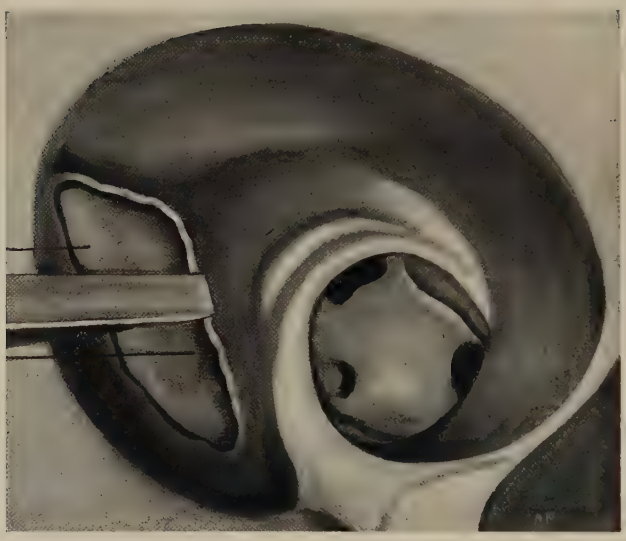

FIG. 79

JANSEN-NEUMANN OPERATION ON THE LABYRINTH

The inner table has been removed over the anterior portion of the lateral sinus and cerebellum, as far forward as the labyrinthine capsule Position of chisel in removing posterior surface of petrous pyramid is indicated

LS—Lateral sinus

C—Cerebellar dura

THE JANSEN-NEUMANN OPERATION

The endocranial *Jansen-Neumann* operation is done as follows; first a complete *Schwartze-Stacke* operation is done. Then by means of a broad flat chisel the inner table over the anterior portion of the lateral sinus is removed (Fig. 79, Plate XXVII). From this point the inner table is removed in a forward direction until the labyrinthine capsule is reached. This exposes a triangular area of the cerebellar dura in front of the lateral sinus. The base of the triangle is directed upward and corresponds to the angle between the middle and posterior fossæ of the skull. The posterior margin of the triangle corresponds to the anterior border of the lateral sinus, and the anterior margin of the triangle corresponds to that portion of the petrous pyramid which contains the labyrinth.

This plate of bone can be removed either by means of a chisel or with a rongeur.

The dura is now carefully separated from that portion of the posterior surface of the petrous pyramid which contains the labyrinth, either with a narrow straight periosteal elevator or with a pair of dressing forceps. This separation must be done very carefully, as the posterior surface of the petrous bone contains numerous small depressions, into which processes of the dura extend. In attempting to separate the dura it is very easy to tear through the membrane at these points.

With a straight chisel placed parallel to the posterior surface of the petrous pyramid, the bone is now removed in thin shavings, the strokes being directed from

the base toward the apex of the petrous pyramid. In this way, that portion of the petrosa containing the semicircular canals is removed. It is very important that the strokes should be parallel to the posterior surface of the pyramid. A deviation of the strokes upward would endanger the superior petrosal sinus. A deviation downward would endanger the jugular bulb. A deviation outward would endanger the facial nerve.

After the first few strokes of the chisel there appears a small curved portion of one of the semicircular canals (Fig. 80, Plate XXVIII). It is on a level with the round window, and a little less than half an inch behind it. This is the convexity of the posterior semicircular canal. On thinning down the bone a little further, there appear two round openings, about a quarter of an inch apart (Fig. 81, Plate XXIX). The lower opening is on a level with the lower margin of the round window, and is the lower limb of the posterior semicircular canal. The upper opening is on a level with the lower part of the oval window, and is the common limb of the superior and posterior canals. A narrow probe passed through this upper opening in a forward direction, enters the vestibule.

Soon a third opening appears, midway between the first two openings, and somewhat more superficial (Fig. 82, Plate XXX). This is the posterior limb of the external semicircular canal. A probe passed through this canal in a forward direction reaches the vestibule very readily. This last canal is now followed up with the chisel-strokes in a forward direction. It becomes oval in shape, then becomes an elongated

PLATE XXVIII.

Fig. 80

Jansen-Neumann Operation

Showing beginning of removal of posterior surface of petrous pyramid

PS—Posterior semicircular canal

PLATE XXIX.

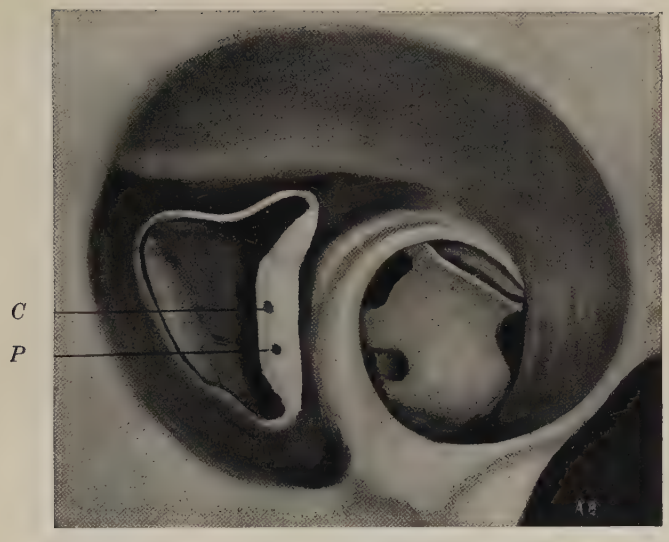

FIG. 81

JANSEN-NEUMANN OPERATION

C—Common limb of superior and posterior semicircular canals

P—Inferior limb of posterior semicircular canal

PLATE XXX.

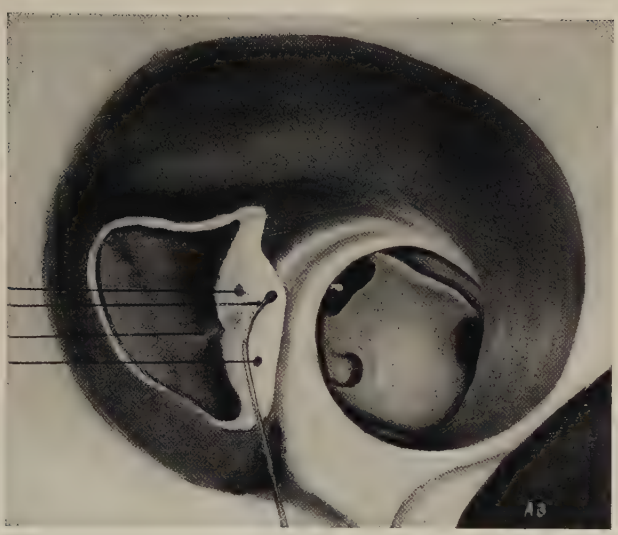

FIG. 82

JANSEN-NEUMANN OPERATION

Probe passed through external semicircular canal into vestibule

C—Common limb of superior and posterior semicircular canals

D—Process of dura going into depression on posterior surface of petrous pyramid

E—Probe in external semicircular canal

P—Inferior limb of posterior semicircular canal

PLATE XXXI.

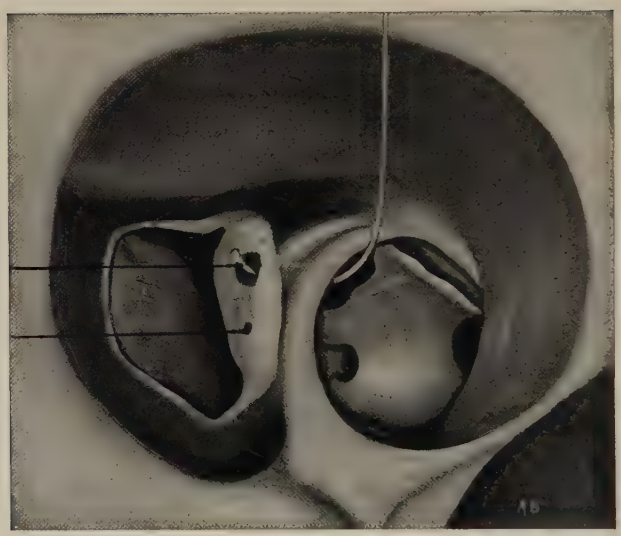

FIG. 83

JANSEN-NEUMANN OPERATION

V—Vestibule containing probe

P—Inferior limb of posterior semicular canal

PLATE XXXII.

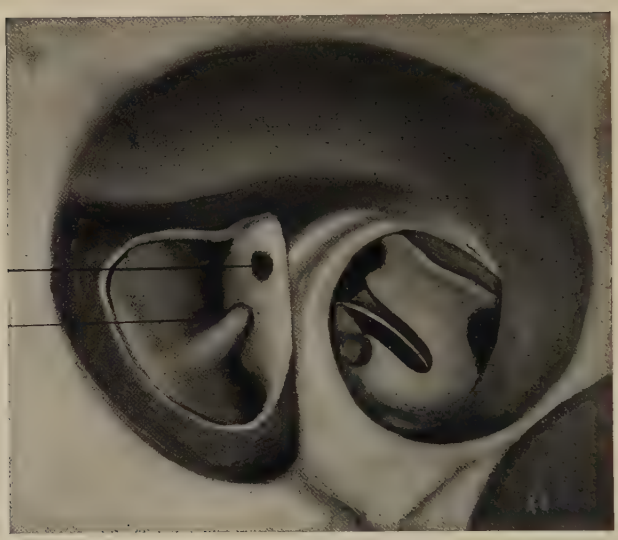

FIG. 84

NEUMANN OPERATION

V—Vestibule

N—Seventh and eighth nerves in internal auditory canal. The lower half of the first turn of the cochlea is exposed

canal, and finally the vestibule appears (Fig. 83, Plate XXXI).

In following up the external semicircular canal, care must be taken to leave its external wall intact. The external semicircular canal is undermined by the chisel, removing only the inner wall of this canal, and the petrosa internal to and in front of it. In this way injury to the facial nerve is readily avoided.

We can assure ourselves that we have really opened the vestibule by passing a bent probe through the oval window in a backward direction. The end of the probe will emerge through the opening in the posterior wall of the vestibule.

At this point of the operation *Jansen* usually stops. *Neumann,* however, continues to remove the petrous pyramid beyond the vestibule until he has removed the posterior wall of the internal auditory canal, and laid bare the seventh and eighth nerves, as they lie in this canal (Fig. 84, Plate XXXII).

Each chip of bone, as it is cut loose with the chisel, should be carefully extracted with a pair of dressing-forceps, care being taken not to tear the dura. If the chip is firmly fastened to the dura at its deeper end, it should be carefully separated with a periosteal elevator. If the dura is accidentally torn, the opening should be enlarged. Only small tears in the dura are dangerous.

The lower half of the basal whorl of the cochlea is then exposed, by chipping off the promontory. This is done with one stroke of the chisel, starting at the oval window, and directing the cut downward and forward.

When the cochlea is a mass of sequestra, pus and

granulations, it can easily be scooped out with a curette.

AFTER-TREATMENT

After doing a radical mastoid, in the presence of a circumscribed labyrinthitis, the posterior wound may be left open, so as to be ready to do a labyrinth operation if a diffuse suppurative labyrinthitis supervene.

If a labyrinth operation has been done, the wound should be left open until all danger of intracranial extension is past, when the posterior wound is closed and a meatal flap made. The flap is made just as in an ordinary radical mastoid operation, and the dressings are done in the usual way.

In cases of labyrinthine empyema, where a labyrinthotomy has been done, the radical cavity heals just as quickly as in an uncomplicated middle ear suppuration. In labyrinthine empyema with superficial paralabyrinthitis, a labyrinthotomy is apt to be followed by a granulating spot on the inner tympanic wall, which lasts for an indefinite time.

When a resection of the labyrinth is done, the rapidity of the healing depends upon the thoroughness of the removal of diseased bone in the petrous pyramid.

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